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<u>OBITUARY</u>

It is with great regret that we report the sudden death at his home of Peter Harrigan, our Honorary Cartoonist. His loss will be keenly felt not only by his family and immediate friends but also by the wide circle of people who have enjoyed his work and have appreciated his representation of people and events in ways they had probably not previously considered.

As members of the South Pacific Underwater Medicine Society we have been greatly favoured by having been given the benefit of his witty and penetrating observations on the world of divers. His cartoons have greatly increased the impact of articles which have appeared in our Journal and they will continue to assist the cause of improving diving safety for many years to come.

The Committee on behalf of all the members of the Society offer their sincere condolences to his wife and family on their loss. He was a nondiver who gave freely of his time to the cause of improving the safety of divers.

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DISCLAIMER

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

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	EDITORIAL	

As Peter Horne comments in his paper on the nitrogen narcosis risks in Cave Diving, the general public considers the perils faced by every diver to be Sharks, Narks and Bends. In respect to the first the risks of attack are very slight, though one fatality did occur in 1985 from what appears to have been a brief excursion into shallow waters by a large deep-water shark. Narcosis is a danger too readily dismissed by divers as something they are too tough to suffer, which is as invalid a belief as a drinker's that his abilities improve after having a few drinks. Failure to recognise the early stages of nitrogen narcosis is a virtual certainty and may be the critical adverse factor involved in fatalities associated with deep dives, the explanation for the omission by some victims of any attempt to ascend when the need for such action should be readily apparent. Dr Pilmanis has suggested another possible factor which could impair the diver's survival chances, carbon dioxide retention. No such cause of deep scuba dive fatality has been noted in Australia or New Zealand, but as neither narcosis nor carbon dioxide as such can be identified at the autopsy, the part they may have played is not capable of proof. Meanwhile, The Bends continues to strike both at those who place too great trust in the degree of safety The Tables can provide and those whose philosophy discounts the possibility of their suffering the indignity of DCS symptoms.

The paper by Dr Straun Sutherland indicates that the real perils posed by marine creatures can be less newsworthy, though probably rather more unpleasant, than a shark attack, as the pain lasts longer. There is very possibly somebody, somewhere, who would be willing to leap to defend the reputation of the stonefish but their gross lack of beauty and a habit of concealment contrasts adversely with the flaunted beauty of the spines of the butterfly cod. Victims feel they have been unjustly "attacked" for a completely unintended contact with the fish. But stonefish never attack, they "merely" activate a mechanical disapproval system when at risk of being trodden on. And what disapproval! In our homocentric way we feel it is unfair that they do not advertise their presence to us. Fortunately, the Commonwealth Serum Laboratories have developed a very effective antivenom and the suggested treatment protocol is given in this paper. As noted in a previous Journal, Dr Sutherland's book is an excellent source of information on Australian

animal toxins.

Decompression sickness is the subject of additional attention in these pages, there being information concerning a new portable chamber, up-to-date details of DES, and details of the treatment teaching, and research centre at the Royal Adelaide Hospital. This is an initiative which has promise of having an important role to play in diving in Australia and overseas. There is also an interesting update on the Beer Bottle talks which have introduced so many divers to the theory of DCS. It is hoped that divers do not need reminding of the increased danger of DCS when suffering fatigue or a hangover. The paper by Dr Robertson is a timely reminder to those who think treatment in a recompression chamber (RCC) guarantees complete cure. They have never understood the moral of the tale of Humpty Dumpty. The 20 per cent of patients who were left with residual disabilities were not the result of poor treatment but of the damage caused before treatment was commenced. Play it safe and remain well within the times "allowed".

In his paper on the Catalina Island Marine Science Centre, Dr Pilmanis covers a multitude of aspects of matters important to divers, from the interaction of regulator efficiency and carbon dioxide retention to an in-depth discussion of the factors involved in saturation and habitat diving. There is a particular appropriateness in the interest being shown in DCS in space. The need for a RCC in an orbiting laboratory not being something most would expect, because Paul Bert's initial work was on altitude, the balloonists of his day being the equivalent of space explorers. He later came down to earth literally, where the importance of his findings was immediately recognised by those involved, either as engineers or doctors, in the art of the sinking of caissons to build bridge foundations and in digging tunnels. There is even now an incomplete understanding of isobaric physiology, so it should be no surprise that diving continues to provide fresh problems, and fresh insights into the reasons for and management of such matters.

The Seasons Greetings are offered to all readers, coupled with a hope that all will contribute in the coming year to the increase in awareness of divingrelated problems and their management, or (better still) their prevention.

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.

MEMBERSHIP

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

The subscription for Full Members is \$A35.00 and for Associate Members is \$A25.00. New Zealand members' subscriptions (\$NZ 45.00 and \$NZ 30.00 respectively) should be sent to Dr P Chapman-Smith, Secretary/Treasurer of the New Zealand Chapter of SPUMS, 67 Maunu Road, Whangerei.

Membership entitles attendance at the Annual Scientific Conferences and receipt of the Journal.

Anyone interested in joining SPUMS should write to the Secretary of SPUMS,

Dr David Davies Suite 6, Killowen House St Anne's Hospital Ellesmere Road MT LAWLEY WA 6050

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

The requirements for the Diploma of Diving and Hyperbaric Medicine are

- 1. To have completed both the introductory course and the advanced course in Underwater Medicine at the Royal Australian Navy School of Underwater Medicine.
- 2. To have completed the course in Hyperbaric Medicine at the Prince Henry Hospital, Little Bay, Sydney, New South Wales.
- 3. To have completed six months full time, or equivalent part time, employment in diving or hyperbaric medicine.
- 4. To present a satisfactory thesis (suitable for publication, usually in the SPUMS Journal) for consideration.

The decision to award the Diploma lies with the Diploma Committee which is comprised of the President of SPUMS, the Officer in Charge of the Royal Australian Navy School of Underwater Medicine and the Director of the Hyperbaric Unit at Prince Henry Hospital.

Applications should be directed to the Secretary of SPUMS,

Dr David Davies Suite 6, Killowen House St Anne's Hospital Ellesmere Road MT LAWLEY WA 6050

SPUMS JOURNAL

Instructions to Authors

Contributions should be typed in double spacing, with wide margins, on one side of the paper. Figures, graphs and photographs should be on separate sheets of paper, clearly marked with the appropriate figure numbers and captions. Figures and graphs should be in a form suitable for direct photographic reproduction. Photographs should be glossy black and white prints at least 150 mm 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: 1766-1770 [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. Page numbers should be inclusive. Examples of the format for journals and books are given below.

- 1 Anderson T. RAN medical officers' training. SPUMS J 1985; 15(2): 19-22.
- 2 Lippmann J, Bugg S. The diving emergency handbook. Melbourne: JL 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, eg. 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.

Reprinting of Articles

Permission to reprint original articles will be granted by the Editor, whose address appears on page 2, provided that an acknowledgment giving the original date of publication in the SPUMS Journal is printed with the article.

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

Airmail Delivery

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

Zone 1	eg. Papua New Guinea, Sth Pacific	\$6.50
Zone 2	eg. Indonesia and Malaysia	\$9.00
Zone 3	eg. India and Japan	\$11.50
Zone 4	eg. USA and Israel	\$14.50
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For further information write to SPUMS 80 Wellington Parade EAST MELBOURNE VIC 3002 Australia

SPUMS JOURNAL BACK NUMBERS

Some copies of a few past issues are available at \$2.00 each including postage.

The relevant issues are

- 1983 Vol. 14, No 3 (4 copies) This contains the 1982 provisional report on diving related deaths in Australia.
- 1984 Vol 14, No 1 (23 copies) This contains Professor Brian Hill's paper on "Decompression Physiology" presented at the 1983 Annual Scientific Meeting.
- 1984 Vol 14, No 2 (20 copies) This contains papers presented at the SPUMS-RAN Meeting in August 1983 and at the ANZICS-SPUMS Meeting in Rockhampton in October 1983.
- 1984 Vol 14, No 3 (18 copies) This contains further papers presented at the ANZICS-SPUMS Meeting in Rockhampton in October 1983.
- 1985 Vol 15, No 4 (23 copies) This contains papers from the 1985 Annual Scientific Meeting in Bandos and from the New Zealand Chapter of SPUMS Meeting in November 1985, including an account of the formation of the New Zealand Chapter.

Orders, with payment, should be sent to

SPUMS 80 Wellington Parade EAST MELBOURNE VIC 3002 Australia

DIVER EMERGENCY SERVICE

008-088-200

The duty supervisor of the Intensive Care Unit at the Royal Adelaide Hospital will answer the telephone and when told that it is a diving emergency will contact the on-call diving doctor. The call will be diverted to the diving doctor who will offer the caller expert advice. Civilian and naval doctors experienced in the treatment of diving accidents from all over Australia will be taking part in DES.

The diving casualty should contact DES on 008-088-200. In most cases he will be advised to attend the local hospital unless he has easy access to one with a hyperbaric unit. That hospital will be contacted by DES with advice. The hospital will notify the nearest hyperbaric unit and arrange a hospital to hospital transfer. It will also notify the local ambulance service. If necessary the hyperbaric unit will alert the retrieval agency, such as the National Safety Council of Australia (Victorian Division) who have portable recompression chambers and aircraft to carry them. If specialist transfer is necessary the local ambulance service will arrange it with the retrieval agency.

ELECTION OF EXECUTIVE COMMITTEE 1987-1988

Nominations will be called for the positions of President, Secretary, Treasurer, Editor and three committee members in December 1986. Each nomination has to be proposed and seconded and countersigned by the nominee.

Nominations are to be in the hands of the Secretary of SPUMS (Dr David Davies, Suite 6, Killowen House, St Anne's Hospital, Ellesmere Road, MOUNT LAWLEY WA 6050) by 1 March 1987.

Those elected will take office at the 1987 Annual General Meeting.

DOCTORS WITH TRAINING IN UNDERWATER MEDICINE

We publish below a list of current members of SPUMS resident in Australia who have completed at least the Royal Australian Navy School of Underwater Medicine introductory course or who have notified the Secretary as requested in the SPUMS Journal (1985 15(2): 3) that they have had equivalent training.

The list has been compiled with the cooperation of the School of Underwater Medicine (SUM) and includes all members that can be identified from the SUM records as well as those who wrote in. As a result it may include doctors who no longer do diving medicals.

The addresses given are those to whom the Journal is sent and so may not be their professional rooms.

Those who have had equivalent training and whose names are not in this list are asked to write to the Secretary of SPUMS giving details of their training.

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Dr P Kolisch 33 Mann Street NAMBUCCA HEADS NSW 2248

Dr R Lloyd-Williams 102 Yanko Road WEST PYMBLE NSW 2073

Dr C Lowrey 233 Raglan Street MOSMAN NSW 2088

Lcdr CJ McDonald PO Box 875 BALLINA NSW 2478

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Dr DB Wallace 1/26 Aubin Street NEUTRAL BAY NSW 2089

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Dr P McCartney PO Box 1317N HOBART TAS 7001

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Dr CBE Davis 8 Ascot Street North BALLARAT VIC 3350

Dr J Knight 80 Wellington Parade EAST MELBOURNE VIC 3002

Dr C Lourey 25 Hastings Street FRANKSTON VIC 3199

Dr JE Mannerheim 25 Wellard Street BOX HILL VIC 3128

Dr I Millar National Safety Council of Australia (Vic Division) 1 Chickerell Street MORWELL VIC 3840

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Dr J Silver PO Box 140 WILLIAMSTOWN VIC 3016

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Dr G Deleuil 135 Dunedin Street MOUNT HAWTHORN WA 6016

Dr H Oxer 331 Riverton Drive SHELLEY WA 6155

Dr J Rippon 764 Canning Highway APPLECROSS WA 6153

Dr A Robertson Sick Quarters HMAS Stirling PO Box 228 GARDEN ISLAND WA 6168

Dr J Taylor PO Box 498 EXMOUTH WA 6707

Dr R Wong 34 Loftus Street NEDLANDS WA 6009

Errors in this list should be notified to the Secretary of SPUMS

Dr David Davies Suite 6 Killowen House St Anne's Hospital Ellesmere Road MT LAWLEY WA 6050

PROJECT STICKYBEAK

This project is an ongoing investigation seeking to document all types and severities of diving-related incidents. 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 organization to increase diving safety through better awareness of critical factors. Information may be sent (in confidence) to:

Dr D Walker PO Box 120 NARRABEEN NSW 2101 REPORT OF THE NEW ZEALAND CHAPTER OF SPUMS TO THE SPUMS EXECUTIVE MEETING OCTOBER 1986 AT ROCKHAMPTON

The New Zealand Chapter has maintained its momentum with its total membership now exceeding 130, an increase of 100 members from the time the chapter was formed.

Dr John Knight was the recognised catalyst for this increase in membership by being our first overseas speaker and his continued excellent contributions to the SPUMS Journal.

The New Zealand 1986 AGM is to be held at Whangamata, 13 -16 November and is already well subscribed. Our Guest Speaker is Dr Des Gorman who will also contribute to a pre-conference meeting at the Philomel Naval Base on the treatment of Diving Accidents with emphasis on Hyperbaric Medicine. Dr Peter Robinson is organising this pre-conference meeting.

Dr Mark Fraundorfer, 1986 SPUMS Conference Organiser, has been appointed Medical Advisor to the New Zealand Underwater Association. SPUMS (NZ) and the NZUA are working in close co-operation on projects of mutual interest. A sport diving medical examination form has recently been prepared by SPUMS (NZ) and approved for circulation and use by the NZUA. The form is reproduced opposite.

Both organizations are concerned at the Diving Accident Rate and are addressing the problem of continuity of treatment facilities on the New Zealand Scene. Three major organisations are involved, Accident Corporation Commission, Health Department and Hospital Boards and the Navy, who at present treat the majority of cases.

SPUMS Executive Members have pursued a policy of increasing General Practitioners' awareness of conditions contraindicating Scuba diving such as Asthma, Epilepsy, Diabetes, Pneumothorax and Pregnancy. An Auckland group of SPUMS members have joined together to form a co-operative called DMAC for the purpose of providing a referral and consultation service. Included in this service is access to Histamine Lung Challenge testing for Asthma (Bronchial Hyperactivity).

Dr Harold Coop has designed a SPUMS (NZ) insignia which is being presented for ratification at our AGM. Dr Peter Chapman-Smith, our enthusiastic Secretary Treasurer, reports he has a healthy credit balance from New Zealand Subscriptions (Full \$45/Associates \$30) which is awaiting the NZ\$ to strengthen before repatriation of funds to Treasurer Dr Grahame Barry. Peter is also proposing to circulate a Newsletter and will be organising the 1987 Conference (Bay of Islands or even Fiji). Dr Mike Davis has agreed to be conference organiser for 1988 at the Marlborough Sound site of the sinking of the Russian Ship Mikhael Lermontov. In addition to Drs Chapman-Smith, Fraundorfer and Davis, I must pay tribute to Andy Veale and Peter Robinson for their extra efforts for Diving Medicine in New Zealand.

The magnitude of the New Zealand diving medical problem, described by Douglas Walker in his case reports and John Knight at our November 1985 Conference, is more serious that at first thought. In the past year there have been 10 diving deaths and 90 divers recompressed.

Page 2.

Page 1.

DIVERS MEDICAL EXAMINATION (SPORTS DIVING) (To be retained by medical examiner)

FULL NAME	DATE//
ADDRESS	DOB//
	Phone No.

OCCUPATION

HISTORY - Have you ever suffe	ered from any of these:
	NO YES
1) Any heart condition	
2) Chest pain	
3) Shortness of breath	
4) Asthma	
5) Other lung condition	
6) Do you smoke	
7) Nose or sinus disease	
Past ear surgery or	
ruptured drum	
9) Dizziness or fainting	
10) Fits or epilepsy	
11) Concussion	
12) Diabetes	
13) Operation or injury	
14) Any other illness or	
disability	
15) Have you ever failed a	
Medical examination	
Any regular medication	
Previous Diving Experience	

I hereby certify that the above information is correct and authorise release of the attached certificate to my Diving Instructor. Further Medical inform-ation may be released to a designated N.Z.U.A. Medical Officer on request. Signed

Date

UALM

EXAM	INATION		Page 3.
Age Heig Weig Puls Bloc PEFF	ght ght se/ d Pressure/	yrs cms kgs 1/m mmHg	
		NORMAL EXAM	l
		YES NO	
1) 2) 3) 4) 5) 6) 7) 8) 9) 10)	Cardiovascular system Respiratory system Effort tolerance External, middle and inner ear Eustachian tube patency Mouth and teeth Abdomen Nervous system Locomotor system Identification marks	y	
in	The following invest some situations:	igations MAY be NORMAL TES YES NO	indicated T
A) B) C) D) E)	Chest X-ray Date// Spirometry // ECG /// Exercise ECG /// Audiogram ///		

COMMENTS

DIVING MEDICAL - Guide to Medical Examiner

1) Diving frequently involves heavy sustained effort. often without opportunity to rest. The diving gear is heavy and awkward to lift and carry on land or boat. Special attention should therefore be paid to the cardiovascular and respiratory systems.

2) All air containing spaces must equalise pressure readily. Special attention should be paid to equalising middle ear pressure (Valsalva manoeuvre), and obstructive lung disease (e.g. history of asthma).

Even momentary impairment of consciousness 3) underwater may result in death (e.g. epilepsy).

4) The following are contraindications to diving: a. Epilepsy

b. Insulin treated Diabetes

c. Asthma

d. History of spontaneous pneumothorax

e. Ischaemic heart disease

f. Pregnancy

5) Other medical conditions which may require special consideration are: Previous middle ear or lung surgery, hypertension, impaired lung function, severe migraine, severe motion sickness, psycholog-ical disorders, physical handicap, visual or hearing impairment, obesity, heavy smoking drug or alcohol use, and advancing age.

Some medical conditions (e.g. recent chest or ear infection, trauma, etc.) may render an applicant temporarily unfit to dive, and will require reassessment at some later date.

Should there be any doubt as to the suitability of a diving applicant please contact the NZUA to be directed to further medical advice.

NZUA P.O. Box 875 AUCKLAND Ph. (09) 895456

A detailed guide for the medical examination of divers may be obtained free of charge from the NZUA, ACC or Health Dept.

	DIVERS MEDICAL CERTIFICATE
FUL Add	L NAME
DAT	E _/_7
Thi ful	s is to certify that the abovementioned has been ly examined by me this day.
1)	He/She has been found to be fit for diving. Advice to Instructor (where relevant)
2)	He/She is permanently unfit for diving.
He/ yrs	She should undergo a routine medical review in
	Séanad

Signed	
Medical Practitioner	
(Print)	
Address	
Ph. No.	
	_

Instructors Stamp

STONEFISH

<u>GENUS SYNANCEIA (LINNAEUS),</u> STONEFISHES: S. VERRUCOSA (BLOCH AND SCHNEIDER) AND S. TRACHYNIS RICHARDSON

Struan Sutherland

'Suppose that fella nail go along your foot, you sing out all a same bullocky all night. Leg belonga you swell up and jump about? Bingie (belly) belonga you, sore-fella. Might you die.'

This description of a Stonefish sting by an Aboriginal first appeared in Confessions of a Beachcomber in 1908.¹ There has been little change! Stonefish are still the most dangerous venomous fish in the world and have a justifiably evil reputation in tropical and subtropical waters.

The names given to Stonefish in general are selfexplanatory: the Devil Fish, the Warty Ghoul and, best of all, Nohu or the Waiting One. Since ancient times the Australian Aboriginal has been aware of the danger of these creatures. Roughley² described how certain north Queensland tribes taught the young to beware of Stonefish as part of the initiation ceremonies, by using a beeswax model of a Stonefish and mimicking the illness produced by its sting.

DISTRIBUTION

Species of Stonefish are found throughout the whole Indo-Pacific region and, in Australia, on the northern coasts from near Brisbane to 500 km north of Perth. Of the two Australian species, the most important is S. trachynis, the Estuarine Stonefish, which inhabits shallow water from Moreton Bay in southern Queensland to as far west as Houtman's Abrolhos Islands in Western Australia. S. verrucosa, the Reef Stonefish, is equally dangerous, but appears to be relatively uncommon.³ S. horrida (Linnaeus), the Indian Stonefish, was at one time incorrectly believed to occur in Australian waters.

DESCRIPTION

Stonefish are so called, because in their natural habitat they are easily mistaken for a piece of rock or dead coral which has become encrusted with marine growth. This camouflage which allows them to snap up any passing prey, makes it almost impossible for swimmers to see and avoid them.

S. trachynis and S. verrucosa, both heavily built fish, are particularly unattractive. Endean³ found the mean standard length of fifty-two specimens of S. trachynis was 20.3 cm and Grant⁴ records a maximum length of 47 cm. Stonefish do not have scales and their soft skin is covered with a variety of tubercles. Cameron et al⁵ reported that the copious secretions from these tubercles contained two fractions (crinotoxins) which were toxic to small marine creatures. These secretions which are bitter to taste, possibly to discourage potential predators, are however, unlikely to cause any injury to man. The small eyes are difficult to see and are carried on bony protrusions. The two species can be distinguished by the fact that the eyes of S. verrucosa are on quite separate protrusions, but those of S. trachynis share the same boss. Moreover, the latter has a yellowish green mouth cavity, while that of S. verrucosa is whitish flecked with red.⁴ Apart from these minor differences, the two species are very similar and, since practically all research work and clinical cases to date involve the commoner S. trachynis, little further reference will be made to S. verrucosa.

Habits

The Stonefish has very large pectoral fins, which allow it rapidly to dredge sand or mud from beneath itself, so that it can settle deeply with only its mouth and eyes fully exposed. From this position, motionless, it can suck a passing fish into its very large mouth, After it has caught a victim, it will rescoop its rest place and settle down again in the sand or mud. The large pectoral fins give the fish a strange, floppy swimming motion, which is best seen in an aquarium tank. In a tank, a captured Stonefish tends to lose a lot of its camouflage due to the activity of the aquarium filter system. Stonefish may tolerate brackish water and, in fact, can survive at least twenty-four hours out of the water, provided they are kept cool and moist.

Venom Apparatus

The venom apparatus of this fish is purely defensive, and plays no role in the capture of its prey.

S. trachynis has thirteen dorsal spines, each of which carries two venom glands. The spines are stoutly built and taper to fine points. Each spine has two lateral grooves, into which the spindle-shaped venom glands are firmly attached by connective tissue, especially Histologically the glands, which are proximally. surrounded by thick fibrous tissue, contain discrete granules of proteinaceous venom. The granules are found both in the tortuous ducts of the gland and secretory cells. The distal portion of the gland becomes modified to form the slender duct, which is actually blocked by fine fibrous tissue stretching across the groove it occupies. Thus, in the normal course of events, there is no apparent way in which the Stonefish can voluntarily expel its venom.

When disturbed, the spines become erect, the anterior and longer first three spines are then nearly vertical while the other spines point backwards at lower The tip of each spine is clearly visible, angles. projecting as it does through a small hole in the sheath of skin covering the spine. Should an object, for example, a human foot, be pressed down on the spine, then, as the spine penetrates the object, the small but tight aperture in the duct is jammed down over the venom gland. As the venom gland is firmly surrounded by fibrous or rigid tissue, liquid venom and venom gland tissue are forced up the duct which has become unblocked. When Endean³ forced rubber sheeting down over the dorsal spines, he described the escape of material from the venom gland as 'a violent



Figure 1. Venom apparatus of a Stonefish:

- A. Dorsal spine viewed from behind, showing paired lateral venom glands (VG). Note tip of spine (S) and narrow venom ducts (VD), both passing through a narrow opening in the tough integumentary sheath (IS).
- B & C A human has trodden upon the fish, the skin is pushed down the spine (large arrows) and the venom gland is compressed by the crumpled sheath. This pressure forces the venom gland to empty up the narrow duct, and venom and glandular tissue spurt (small arrows) into the deep tissue of the foot.

expulsion'. He demonstrated that pressure directed upon the venom glands did not release venom. Wiener⁶ on the other hand, considered great care had to be taken to avoid pressure on the glands when they were being dissected out, otherwise venom was easily lost. Venom liberated by the sheath being forced over the gland contained a mixture of granules and venom gland cells, either singly or in groups. Endean considered that regeneration of both venom gland and venom stores took some three weeks.

Endean recorded that, in an average sized fish (20 cm in length), .the venom glands are 1 cm in length and 2 mm in diameter. The venom duct is some 7 mm in length. He estimated that the depth of penetration of the spine required to cause venom release would be 0.6 cm in the case of a small fish to 1.0 cm with a mature fish. Endean stressed that even a small Stonefish 7.8 cm long had well developed spines and venom glands.

Venom

Duhig and Jones,⁷ who pioneered studies on Stonefish venom, showed the venom remained toxic in the venom glands for several days after the death of the

fish. They found that the venom was haemolytic in vitro to a variety of erythrocytes, but only in the absence of serum. This effect is not considered of clinical significance.

Wiener⁶ dissected out the venom glands from living specimens of S. trachynis and then freeze dried the venom he collected. He found that as much as 0.03 ml could be aspirated from a single venom gland. Dissection of seven specimens, of average length 26 cm and average weight 0.7 kg, yielded an average of 6 mg of venom per spine. The total yield of venom per fish ranged from 49 mg to 88 mg. Wiener noted that often the venom glands were not full and sometimes the gland had been replaced, possibly with strands of connective tissue.

Wiener also studied the toxicity of S. trachynis in a number of animals. He resuspended the venom in saline buffered at 7.4 and then centrifuged it to remove cellular material and debris. He estimated that 25 to 30 per cent of the harvested material was lost in this way, but decided to ignore this loss when estimating toxicities which related directly to the starting material. Thus, the quantities of venom injected are 25 to 30 per cent lower than stated. In mice of 15 g weight the LD50 of venom was 0.004 mg intracerebrally, 0.007 mg intravenously, 0.03 mg intraperitoneally and 0.05 mg subcutaneously. The figure for the intravenous LD50 compares favourably with that of 0.004 mg obtained by Saunders⁸ using venom freshly extracted from S. verrucosa.

In guinea pigs of 250g body weight, a subcutaneous dose of 4 mg of venom caused death within three hours. Death was preceded by weakness and coma. A guinea pig dosed with 2 mg died on the third day. Oedema and necrosis had developed at the site of the injection. Guinea pigs receiving 1 mg or 0.6 mg died on the sixth and eleventh day respectively. A dose of 0.2 mg caused a skin lesion only. Post mortem studies on these animals was not instructive.

Wiener determined that the minimum ischaemic dose of venom (intradermal) in a rabbit was 0.002 mg in a volume of 0.05 ml. When he gave an intravenous injection of venom (0.17 mg/kg) into an anaesthetized, monitored dog, transient hypertension and apnoea occurred. Larger doses produced cardiac and respiratory arrest within two minutes. Saunders⁸ found that, when S. verrucosa was injected intravenously into monitored rabbits, hypotension developed which was associated with electrocardiographic changes such as atrioventricular block and ventricular fibrillation. Saunders⁹ found .also that the actions and toxicity of S. horrida venom are similar to S. verrucosa venom.

Austin et al¹⁰ presumably using S. trachynis venom rather than S. horrida, found that the venom when injected intravenously in rabbits caused hypotension, respiration distress and muscular paralysis. These studies suggested that Stonefish venom has powerful myotoxic properties which can cause loss of function of all muscle types. The paralysis appeared to be caused by a conduction block in the muscle due to slow polarisation. They considered that the death of the rabbits was due to paralysis of the diaphragm, rather than myotoxic effects on the heart muscle.

Austin et al¹⁰ undertook some biochemical studies upon the venoms of S. trachynis and S. verrucosa. They found that the major toxin of both is a labile protein with a molecular weight of the order of 150 000. The venom was shown to contain a potent hyaluronidase and a capillary permeability factor. No protease or phospholipase A activity could be found, the venom had no effect on blood clotting, nor did it cause haemolysis. Deakins and Saunders¹¹ investigated methods of isolating the toxin and found starch/gel electrophoresis to be a suitable procedure. They also found the toxin to be labile, but it could be stabilized for some months if lyophilized with 5 per cent sucrose.

HUMAN ENVENOMATION

Stonefish stings are extremely painful. The pain is simultaneous to penetration by the spine and rapidly worsens. Swelling of the envenomed area develops at the same time and may soon extend. The victim may be irrational because of the pain. The severity of the signs and symptoms is usually in direct proportion to the depth of penetration of the spine(s) and the number of spines involved.

As well as the local effects, muscle weakness and paralysis develop in the affected limb and varying degrees of shock may occur. Human fatalities have been caused by Stonefish stings in the Indo-Pacific regions, but no known deaths have occurred near Australian waters.

Smith¹² described two deaths due to S. verrucosa. One occurred in the Seychelles in March 1956, when a healthy 15-year-old boy collapsed became cyanotic and died within a short time(not defined) of receiving the punctures to his foot. He suffered terrible agony before he became unconscious. The other fatality involved an adult man who died within an hour of standing on a Stonefish at Pinda, Mozambique. He collapsed and was 'almost delirious' prior to lapsing into unconsciousness.

CHOICE OF ANTIVENOM

Stonefish antivenom will neutralize the venoms of S. trachynis, S. verrucosa and S. horrida. It is prepared by immunizing horses with the venom of S. trachynis and is available as a pure equine $F(ab)_2$ preparation. Ampoules contain approximately 2 ml, which will in vitro neutralise 20 mg of venom.

Miss Wendy Cowling kindly prepared the following brief survey of reports of Stonefish antivenom usage over the period 1965 to 1981 (inclusive).

A total of 267 reports were received at the Commonwealth Serum Laboratories from the following sources: Queensland 129, Northern Territory 35, Western Australia 54 and overseas 49. Males were stung in 83 per cent of cases and, apart from three persons injured on their arms or wrists, all stings occurred either on the hands (36 per cent) or the feet (63 per cent). The victim's average age was 27 years and the median age was 25 years. Four children under the age of 5 years received antivenom for stings. No deaths were reported.

Sutherland and Trinca¹³ published details of Stonefish stings which occurred specifically in Queensland from 1967 to 1978. In the period considered, ninety-five Queensland cases received antivenom and eightyseven of the cases involved males. Injuries were restricted to the hands or feet twenty-three of the former and seventy-two of the latter. The seasonal incidence was as follows: January (7), February (3), March (12), April (12), May (8), June (6), July (7), August (10), September (10), October (2), November (7), December (11).

The higher rate of stings amongst males compared with females probably reflects the men's professional or amateur fishing activities. The high incidence in March/April and August/September may be related to Easter and school holidays. The limitation of injuries to either the hands or the feet suggests that many of these stings could have been avoided with appropriate care.

FIRST AID

No attempt to should be made to retard the movement of venom from the stung area. To delay the escape of venom will only enhance local pain and tissue damage. Relief of pain is the most urgent requirement. In minor cases this may be achieved by bathing the stung area in warm to hot water (but not scalding the skin). However, hot water is not often available. In severe cases. pain relief may only be obtained by the combined use of antivenom and potent opiate drugs, such as morphine or pethidine.

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CLINICAL MANAGEMENT

Local Anaesthetic As Additional First Aid

It is often recommended that a local anaesthetic, such as lignocaine or the longer acting bupivacaine, be injected into the track of the sting and the surrounding area. Such a procedure may afford pain relief for sixty minutes or so, but should only be considered as an adjunct to the administration of antivenom. Mixtures of adrenaline and lignocaine should never be used to provide local pain relief, since adrenaline will decrease the blood flow in the region.

Very rarely, when there are multiple stings and the agonized victim is almost irrational and uncontrollable, the use of a regional block, which is easily performed by an anaesthetist, should be considered. A general anaesthetic is not recommended. A regional block, if bupivacaine is used, has the advantage of lasting eight to twelve hours and so will not enhance the systemic effects of the venom and be much safer for the patient than a general anaesthetic.

Other Local Measures

Incision of the stung area. Quite often incisions are made into the stung area to promote bleeding and, hopefully, the escape of venom. The writer does not usually advise such measures, but accepts the fact that, in very severe cases, if they are performed professionally, the clinical outcome may be improved.

Emetine. Injection of emetine hydrochloride (65 m/ ml) into and around the wound has produced some relief of pain, probably due to the acidity of the solution inactivating the venom. However, there are now few commercial sources of emetine hydrochloride.

Potassium permanganate (Condy's Crystals). This solution should not be used, as it will merely cause local tissue damage and aggravate the wound. It is probable that, as was shown with trypsin in snake bite cases,¹⁴ the potassium permanganate cannot effectively pursue the injected venom through the tissues.

Indications For Antivenom

Antivenom is recommended for all cases, except those involving only a puncture wound with moderate discomfort. Other exceptions, which may not need antivenom, are those cases reaching medical care (and antivenom) a number of hours after the sting, which are not severe stingings and which are clinically improving.

The initial dose of antivenom will depend upon how many spines have deeply punctured the skin, for example:

- 1 or 2 punctures, contents of one ampoule (2,000 units)
- 3 or 4 punctures, contents of two ampoules (4,000 units)
- 5 or 6 punctures, contents of three ampoules (6,000 units)

Each ampoule contains 2 ml of antivenom. The antivenom is usually given intramuscularly. It is becoming acceptable practice to administer antihistamine, adrenaline and sometimes steroids prior to the injection or infusion of antivenom. Once the decision to give antivenom has been made, proceed as follows:

- 1. Give the patient an appropriate dose of antihistamine parenterally.
- 2. Draw up in a syringe 1 ml of adrenaline (1 mg/ml). If the patient has a known allergy to equine protein or has had antivenom before, give a small dose of adrenaline subcutaneously (for example, 0.5 mg for an adult). It may be wise to give all other patients 0.25 mg of adrenaline subcutaneously as a prophylactic measure. A child should be given a smaller dose.
- 3. If the patient has a history of allergy, especially to equine protein, or has received equine protein before, a steroid (eg. 100 mg methylprednisolone) should be given intravenously.
- 4. Antivenom should only be given if full and tested facilities are available for treating an anaphylactic reaction.
- 5. Fifteen minutes after the parenteral administration of the antihistamine (with or without adrenaline at another site), give the antivenom intramuscularly.

In a very severe case, three or more ampoules may be required and the use of the intravenous route should be considered, particularly if the pain is widespread or the patient is shocked.

Antivenom should not be injected in or around the area of the sting. Such an injection may worsen the local effects of the venom.

Hospital Management

Bed rest is usually essential. The injured limb should be comfortably immobilized and the envenomed region lightly covered with sterile dressings. Like other penetrating marine stings, Stonefish stings are potentially contaminated with bacteria. The likelihood of tissue necrosis caused by the venom renders the injury more prone to clostridial infections than simpler wounds. Therefore, tetanus prophylaxis should be carried out in accordance with the victim's immune status, while .antibiotics may be used to control wound infection. The more severe injuries can require early surgical debridement of dead tissue and drainage. When antivenom has been delayed and considerable ulceration exists, then skin grafting may be necessary.

SEQUELAE

Sometimes a Stonefish sting remains painful for months and/or a recurrent inflammation or discharge occurs at the site of the sting. This is usually due to a foreign body in the wound, in particular a portion of a Stonefish spine. Being semi-transparent and quite deeply embedded, it is easy to overlook a broken-off spine and, if any doubt exists, it is preferable to explore the wound surgically during the initial admission to hospital.

Sometimes no foreign body is found. In one such case a middle-aged Melbourne man suffered a possible Stonefish sting to his skin on a remote island in New Caledonia in 1975. His envenomed leg became grossly swollen and for several days he was delirious and his companions thought he was dying. When he reached civilization several weeks later, the skin was still swollen and discoloured. Over the next three years he suffered episodes of tender reddening of the area, associated with high fever sweating and confusion. These attacks occurred every two or three months and lasted several days. In 1978, he was diagnosed as suffering from recurrent haemolytic streptococcal cellulitis. He responded rapidly to a course of intravenous crystalline penicillin, followed by procain penicillin, and in 1981 he was still free of any recurrence. Because of his response to penicillin and negative x-ray, it was decided not to explore the region of the sting.

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The above is an edited extract from Australian Animal Toxins by Dr SK Sutherland, published by Oxford University Press, covering one of the topics discussed by Dr Sutherland as guest lecturer at the 1985 Annual Scientific Meeting of SPUMS.

Dr Sutherland has copies of Australian Animal Toxins available for \$45.00, which is a large reduction on the bookshop price.

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SPUMS ANNUAL SCIENTIFIC MEETING 1986

THE ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT

Des Gorman

INTRODUCTION

The Hyperbaric Medicine Unit created within Royal Adelaide Hospital (RAH HMU) is a joint venture of the Institute of Medical and Veterinary Science, Royal Adelaide Hospital, and the Victorian Division of the National Safety Council of Australia. The RAH HMU is part of the Department of Anaesthesia and Intensive Care, answerable through the Senior Director of that department to the hospital Medical Superintendent and Chief Executive Officer. It is envisaged that the position of Senior Director will become a professorial appointment to Adelaide University in the immediate future.

ROLES AND FUNCTIONS

The RAH HMU has clinical and education roles, considerable research commitment and the task of co-ordinating the Diving Emergency Service. The

Royal Adelaide Hospital was chosen as the ideal site for the HMU because the hospital is co-located with the Institute of Medical and Veterinary Science and Adelaide University, an excellent retrieval system was already based on the RAH Intensive Care Unit, and because the local abalone fishermen constitute a dysbaric problem of considerable magnitude.

The RAH HMU will provide emergency and nonemergency clinical services to both South Australia and the Northern Territory, and will encompass all aspects of hyperbaric medicine. Clinical services will be based on the Intensive Care Unit, involving Intensive Care Unit registrars and intensive care trained nurses also trained in hyperbaric medicine to act as recompression chamber attendants.

The static recompression chamber installed at RAH was built by DRAGER Pty Ltd, is compatible with NATO bayonet transportable recompression chambers, and

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has been interfaced with all necessary intensive care facilities and equipment. In particular mechanical ventilation and invasive and non-invasive monitoring are possible.

RESEARCH

The RAN HMU will undertake both human and animal model research. Animal model research will be conducted within a dedicated hyperbaric animal laboratory in the Institute of Medical and Veterinary Science, and will include rabbit open brain model studies of cerebral arterial gas embolism and Aplysia identified neuron studies.

Both animal model studies will use a purpose-built animal recompression chamber.

The overall aim of the rabbit open brain studies is to develop an ideal regimen for cerebral arterial gas embolism (CAGE).

Using the invertebrate marine animal Aplysia, identified neurones will be fixed with electrodes and used to study the direct action of oxygen, carbon monoxide, hydrogen sulphide, and cyanide on neurone cell membranes, and in particular an ion channels. The aim of these studies is to identify the cellular site of these toxicities.

A human laboratory is being built for the Department of Anaesthesia and Intensive Care and will be used by the RAH HMU in addition to other divisions of the Department.

The RAH HMU is also initiating many prospective clinical studies which include the treatment of dysbaric illness, traumatic brain and spinal cord injury, cerebrovascular accidents, peptic ulceration, ischaemic and diabetic ulcers.

TEACHING

The RAH HMU will offer training for medical students and practitioners, nurses, and paramedical personnel.

Short duration courses will be available at least yearly for any medical practitioner at two different levels: instruction regarding the assessment of candidates fitness for diving and other hyperbaric exposures, and instruction regarding the recognition and management of dysbaric and non-dysbaric illness suitable for hyperbaric treatment. Course fees will be designed to cover the cost of conducting the course.

Professional training will be provided to both preclinical and clinical medical students, and to registrars engaged in training programmes in medicine, anaesthesia, intensive care, occupational medicine, and hyperbaric medicine as a dedicated specialty.

Medical practitioners and biomedical scientists wishing to undertake supervised research degrees will also be accommodated within the RAH HMU, in either clinical, applied human, or animal model research.

The HMU will also conduct short duration courses for recompression chamber attendants, hyperbaric nurses, recompression chamber operators, and divers.

THE DIVERS EMERGENCY SERVICE

The Divers Emergency Service (DES) is a telephoneadvice service available to Australian and South Pacific divers in the event of diving accidents.

A similar system, the Divers Accident Network (DAN), has operated in the United States of America for several years. The needs of the Australian diving community are different to those of the North Americans (and Europeans), because of the greater percentage of the Australian community that engages in recreational diving, and the greater distance between individual hyperbaric facilities in Australia.

The DES began as an Australian Underwater Federation (AUF) initiative during 1984. At that time the Minister of Defence committed the Royal Australian Navy School of Underwater Medicine at HMAS PENGUIN to provide a 24 hour contact point for diving accident advice.

The initial DES was not toll-free, and became largely restricted to metropolitan Sydney. In addition, the medical practitioners providing the service were restricted to members of the Royal Australian Navy (RAN) serving at the School of Underwater Medicine.

Subsequent modifications to the DES, performed with the endorsement of the Federal Ministers of Defence and Health, have overcome many of these problems by providing a toll-free emergency telephone service. This is attended 24 hours a day by personnel expert in medical retrieval and intensive care, and gives divers access to medical experts throughout Australia. The panel of DES consultants includes both publicly and privately employed medical practitioners, and serving members of the Royal Australian Navy.

The modified DES is based at the Royal Adelaide Hospital Hyperbaric Medicine Unit which provides the toll-free telephone. Telephone calls to the toll-free number (008 088 200), will be answered by the Intensive Care Unit Supervisor and diverted to the duty DES consultant. The caller and the duty consultant may be in different States, neither of which may be South Australia. The DES is not intended to replace or compete with existing resources, and will always direct casualties to appropriate local health facilities. The advice given on diving casualty management will include the likely diagnosis and necessary first-aid measures, as well as necessary local retrieval procedures.

Medical retrieval networks in each State are coordinated by the local ambulance organisations. In general, retrieval of diving casualties will consist of hospital to hospital transfers, and unless the casualty is within a short distance of a hyperbaric unit, they will be diverted to the nearest local hospital for necessary treatment prior to retrieval.

When diving casualties, requiring recompression, are remote from a hyperbaric unit, road ambulance transport will be unacceptable when distances are great, or when the road transport involves decompression of the casualty to 300 m or more above sea-level. Under these circumstances, pressurised (and preferably hyperbaric) retrieval by aircraft is indicated. The Victorian Division of the National Safety Council of Australia (NSCA) operates a hyperbaric retrieval service in most regions of Australia. This unique service uses transportable recompression chambers and includes the necessary personnel and aircraft. It solves the problems caused by the distance between individual hyperbaric facilities. In addition, the service is provided to the South Pacific region.

It is important to emphasise that the DES cannot task the NSCA directly, and that this will always be the responsibility of regional ambulance organisations. The overall service available to Australian diving casualties consists of: the DES as an emergency advisory body; the various ambulance organisations which will control and co-ordinate the retrievals; the NSCA hyperbaric retrieval service acting at the direction of the ambulance organisations; and the various hyperbaric units capable of definitive treatment.

These combined resources will significantly reduce the morbidity and mortality arising from diving accidents, and will produce considerable cost savings to State and Federal budgets. This is illustrated by a recent hyperbaric retrieval of an abalone diver with arterial gas embolism from Portland, Victoria, to Royal Adelaide Hospital. In the absence of this retrieval, which involved a transportable recompression chamber, the delay before treatment would have made survival without major handicap impossible. The cost to the community arising from such morbidity would include that of maintaining a handicapped individual and his family, in addition to the loss of taxation revenue from his former earnings. Given that the diver has returned to work without disability, the cost-savings calculated from this retrieval are sufficient to operate the retrieval system throughout Australia for many years.

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THE UNIVERSITY OF SOUTHERN CALIFORNIA CATALINA ISLAND MARINE SCIENCE CENTER, AND SOME RECENT PROJECTS

Andrew Pilmanis

CATALINA ISLAND

The University of Southern California (USC) is not State supported. USC is a private institution. Although it is Los Angeles it is in no way associated with UCLA except as a rival.

Catalina Island is 20 miles off-shore. It is about 25 miles long. The Institute for Marine and Coastal Studies is located at the west end at Two Harbours or the Isthmus. The Marine Science Centre (MSC) is in a small cove called Fisherman's Cove which has some unique features which is the reason it was placed there.

It was originally to be run by a consortium of universities in Southern California, however the other universities backed out and left the USC with it. It was a financial drain and so a problem in that respect. The plus is that it has been in operation since 1967 and we have had some fascinating projects there.

The Fisherman's Cove area has similar water conditions to Cooks Bay in Moorea except it is somewhat cooler. It is on the east side of the Catalina Island and there is excellent diving. Our average visibility is about 15m but at times it gets to over 30m visibility. The kelp, macrocystus, is the primary attraction. Unfortunately the warm water conditions of El Nino a few years ago totally wiped out the kelp at Catalina. It is just starting to grow back now that the water has cooled off. Kelp requires cool water and we have had almost a 10° C increase, on the average, during the El Nino, which brought warm water up from the South American coast. The warm water conditions lasted about three years. We had actually tropical fish around Catalina.

In the summer the area becomes the local watering hole for the yachting community. There are an enormous number of pleasure yachts in Southern California. Most of these leave their marina once or twice a year and come over to Catalina. The owners go to the bar for a drink or two then go back on the boat and back to the marina on the mainland. It is a yearly ritual.

Movie companies have had a lot to do with the ecology of Catalina Island. For the filming of "The Vanishing Herd" buffalo were brought to the island and 15 buffalo were left on the island. Now there are over 600. They are not very intelligent beasts, they tend to stomp on everything. They are not a hazard but they leave their calling cards on your front door. They have been known to walk up and down the halls of the dormitories which upsets the students. The Isthmus area was used for the filming of the original "Mutiny on the Bounty" in the 1930s with Charles Laughton. The movie company planted exotic trees including many coconut palms for the movie, and they still flourish.

MARINE SCIENCE CENTER

The Marine Science Center was built in 1967. It consists of a laboratory building, dormitory housing and on the waterfront there is a pier, a heliport and the chamber in a large building. There is a marine railway leading to the large building where the chamber is (Figures 1, 2 and 3).



Figure 1. Inside the chamber.

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The heliport pad was put in because of the chamber. For every chamber case that we receive, we have three or four helicopter landings, bringing the patient in and taking the patient out, bringing the physician in and out.



Figure 2. Aerial view of the Marine Science Center, Catalina Island.

The marine railway is a very inexpensive way to launch and retrieve submersibles. The greatest cost when using a submersible is the support vessel, not the submersible itself. The marine railway bypasses the support vessel and allows us to launch and retrieve at will. We have had six or seven different submersibles utilise the facility. At times we have been down to a thousand feet chasing crabs with manipulators on submersibles.

The Marine Science Center is used because there is easy access to very calm, clear waters for a variety of marine science subjects and research and teaching. It is an unpolluted area.



Figure 3. The Catalina hyperbaric chamber

We get marine mammals at Catalina Island including grey whales about 50 or 60 feet long. Sharks are also seen, although up the coast in the San Francisco area we get a fair number of great whites. During the El Nino condition there were 6 or 8 very serious attacks in the San Francisco area. As a result almost nobody went in the water. You could go out in the boat almost any day and find a great white.

Most of our diving is done from small vessels. The University does run large oceanographic vessels which tend to stay away from shore. We have had a number of rather interesting visitors to the Marine Science Center. The Cousteau team did their first TV special on squid at Catalina. The Cove is a squid breeding ground. Cousteau took the Calypso in there and the ship's water intakes got plugged with squid, and everything was over-heating. So they jumped overboard to clear the intakes, found the squid mating and made a TV special out of it.

PHYSIOLOGICAL STUDIES

We will now talk about the specific medical physiological problems that our programs have been associated with over the years. I started off in the space program studying cardiovascular dynamics associated with weightlessness which occurs acutely both in space and underwater. The space program also has chronic problems associated with weightlessness.

Then I went into breathhold diving physiology. We did central venous catheterization on people who then did breathhold dives. The central venous pressure goes up and the heart rate goes down. The bradycardia associated with breathhold diving can reach 4.5 seconds between beats. Inspite of the dramatic bradycardia there was no noticeable effect on the individual during the dive.

Similar cardiovascular changes during breathhold diving occur in marine mammals. We trained a Californian Sea Lion to dive on command therefore it was a voluntary dive in the ocean. Most of the work before that had been done by strapping a sea lion to a board and shoving his head underwater and reporting the various responses. The results of both techniques were very similar in the shifting fluids, the bradycardia, the tremendous vasoconstriction that occurs with the diving reflex. There really was not much difference between the voluntary and the forced immersion.

We got interested in people again and in deeper diving. We did deep dives and studied the cardiovascular responses. I would have to say I would not repeat those dives knowing what I know now but it was interesting at the time. We had underwater ergometers to measure work done underwater. A series of underwater reporting devices were strapped onto the divers for measuring a variety of parameters. We could measure anything we wanted, however primarily we were interested in cardio-vascular responses. We got into respiratory gas sampling during different types of diving and different levels of exercise. We could take at least six gas samples during a dive.

Some of our data is pertinent to sport diving. CO2 retention does occur with scuba diving. Most people feel that since it is an open circuit device that one should not get any CO_2 retention. Well we did. There are three factors that affect the PCO₂ of a scuba diver, the depth which of course governs the density of the

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gas, the level of exercise and the level of resistance in the regulator. There is often a tremendous amount of resistance in the regulator. The mouthpiece opening that one breathes through is only small so divers at depth, working hard do retain CO_2 . At times it was to levels that were of concern to us. We usually found an increase in PCO₂ with depth but we also found a decrease in work capacity with depth. The maximum work that the diver could accomplish, decreases and CO₂ levels rise leading to not very productive diving. We attempted to go deeper than 30 metres and found that we just could not, with good conscience, make those experimental working dives. They were becoming dangerous from the risk of the subjects passing out from CO₂ retention. A tight fitting wet suit is one of the factors that affects respiration, but the density of the gas, the resistance of the regulator and the level of work predominate.

Another thing that had been found a number of times before, in the US Navy, in the Israeli Navy and other places, is that experienced divers tend to have a very low sensitivity to CO_2 . The increase in minute volume with rising inspired CO_2 is a measure of CO_2 sensitivity. Normal people do not retain CO₂ when inspiring a raised CO₂. They increase their minute volume, to maintain a normal PCO₂. Some experienced divers do not increase their minute volume as much, in other words they are less sensitive to CO_2 , and they retain $\rm CO_2$ raising their $\rm PCO_2$. $\rm CO_2$ retention can send you unconscious and the problem is, that you get no warning. One moment you are conscious and then you are unconscious with nothing in between. It is a hazardous situation. I know of two deaths of extremely experienced divers in deeper depths working hard where they simply passed out for no known reason. There was no struggle. There was plenty of air in the tank. No cause other than CO₂ retention could be found. Of course CO₂ diffuses out so fast you cannot prove that it had occurred, but it is very tempting to point the finger at those two deaths as CO₂ retention. With our subjects at high levels of CO, they routinely got headaches. The headaches typically would occur either underwater or immediately after the dive and would last either a few minutes or an hour or two. High PCO₂ was always associated with headaches. We still do not know why some divers retain CO_2 . The last paper from the Israeli Navy more or less left it hanging. It is either a natural selection process, because we only had very experienced divers down, they were not recruits. Or it is an adaptation of some sort. These people are insensitive to CO₂ on the surface as well as underwater.

The resistance in the regulators is a very important aspect. We bench tested every regulator for every dive before and after, and they varied tremendously. In the end we used US Divers Conshelf regulators. We found them to have the least resistance. These had only a few cm of water inhalation and exhalation resistance. The normal regulator would be double, triple or quadruple these numbers. I would be extremely surprised if any of the audience's regulators were in the range we used. It is very difficult to obtain such low resistances. Our data was collected using the low resistance regulators. If one doubles or triples the resistance it makes the situation that much worse.

The divers definitely did hypoventilate relative to their topside respiration. Whether it is the result of using a regulator or is physiological, I cannot say but it was a constant finding.

We are now interested in thermal aspects in divers and in thermal protection. There is a gadget on the market called a heat flow disc. Put a number of discs in strategic areas on the body and you can measure the flow of heat across those discs and then we can come up with total body heat loss, or gain. This is a much better measure of what is really going on in thermal stress than rectal temperature which has been the usual measurement. Using heat flow discs you can obtain information on the actual heat loss. If you just measure rectal temperature on a dive there is a lag before any temperature drop occurs, so although heat loss is occurring it is not being recorded at first. What is much more interesting is the whole body heat loss in watts per metre.² We studied this in a variety of different wetsuits. As one would expect there was greater heat loss when wearing the thinner wetsuits than the thicker. We found that there is an immediate heat loss reading when the diver first jumps into the water and then heat loss generally stabilizes at some level, particularly with the thicker suits. In the thin suits the subjects simply did not last long enough to stabilise as they aborted the dive because they were cold. With rectal temperature, skin temperature, heat flow and water temperature, one can obtain total body heat loss which is what we are interested in. One can calculate the insulation of the wet suit according to the depth, taking into account the changing water temperature (thermoclines) during the dive.

Another project that lasted about four years was with the Doppler bubble detector, for detecting bubbles after a dive or during a dive. An ultrasound transmitter and sensor is focussed on the right heart. It works quite well. I think it is probably responsible for the shift in the last 15 years in how we view decompression sickness, decompression tables and treatment, etc. It is very easy to use. It is not so easy to interpret. One gets a variety of sounds coming out of the right heart. If there is extensive bubbling anybody can pick it listening through the head set. It is the minor to small amounts of bubbling that become a problem. Of course that is generally the area we are most interested in. If somebody comes up with decompression sickness we put a Doppler on immediately, we hear an almost continuous flow of air through the right heart. We cannot hear any of the other heart sounds. The use of Doppler monitoring was started back in the early 1970s by Merryl Spencer in Seattle. He classified the bubble detection as grade 1, 2, 3 and 4, each with increasing numbers of bubbles. It is not very useful because the person listening is the key factor. I remember one of Merryl's number one bubble listeners coming down and listening to one of our subjects. I listened to the Doppler and he listened to it. We came to totally opposite conclusions. At that time I had had four years of Doppler monitoring so there is a tremendous amount of individual interpretation. I think its value is very limited and that people often make too much of the data from Doppler studies. It is useful so long as you realise that who interprets the data is extremely important.

We went through a series of electronic developments in the early 1970s trying to get an electronic signature of a bubble. Unfortunately frequency alone will not do it. One needs several parameters and our laboratory could not develop a satisfactory instrument. We used frequencies, the problem is that each individual and each location has different frequencies. Doppler monitoring is being used extensively now in space programs for monitoring decompression and several other sectors. We continued with Doppler studies both in people and in animals, of particular interest was the effect of exercise on decompression. We used dogs in that study. The conclusion there was that two factors affect the results of exercise during decompression. On the one side the situation is made worse by cavitation effects, mechanical effects initiating bubble formation. On the other side of the coin was an extreme vasodilation and an increased blood flow. One should be eliminating nitrogen faster. As it turned out, at least from this study, the cavitation effect predominated. So it is not recommended to exercise during decompression. It is an area that I think needs a great deal more research.

We also used people in testing the Edge decompression computer with a variety of dive profiles. We monitored them with a Doppler and found no bubbles at all on the profiles produced by the Edge. I find the theory behind the Edge is excellent but nevertheless it is a gadget. I took it out yesterday on a dive, turned it on underwater and it flashed at me that I had forgotten to change the battery and it refused to co-operate. It still comes back to individual responsibility.

HYPERBARIC FACILITY

The chamber obtained in 1974 is large. It was given to us by the Lockheed Corporation because they needed the space for something else. It is not an easy thing to transport around especially across 20 miles of ocean, as it weighs 22 tons. The size is very useful as we have had up to four patients simultaneously in there with a tender each. One cannot manage that in a 54" diameter chamber. The chamber also serves very well for research purposes and for training. We have, in 12 years, made over 2,000 dives in the chamber for training purposes. We can put up 10 people in it at a time. We have done 515 treatments, the last one was the day I left to come to Moorea. It was a woman with a beautiful air embolism who made a good recovery.

UNDERWATER HABITAT PROGRAMME

A very brief history of saturation diving. The concept was developed by Captain George Bond of the US Navy in the 1950s, and we ended up naming our habitat for George Bond. He died about three years ago, a very disappointed man because saturation diving had not developed the way he envisaged it would. The first open water saturation dive, off Southern France, was run by Ed Link in 1962. The diver was Robert Stenuit. Very shortly after that Cousteau put his houses down, several models, and they lived underwater. The interesting thing here is that if one compares Cousteau's Conshelf habitats of 1962 and 1963 with the single habitat still being used there is really very little difference.

This project I started in the early 1970s as far as thinking about it. I actually started in 1980 when we applied to NOAA (The National Oceanic and Atmospheric Administration) a sort of a wet NASA although not nearly as well funded. It was a joint operation between NOAA and the University of Southern California. The purpose was to utilise saturation diving techniques to do marine science in the ocean. This was not anything new. It has been done before by using a number of habitats but generally it has been done in tropical waters not in temperate waters. Our water temperatures, down to 30 metres, varies anywhere from 10° to 20° C.

Predominantly it is in the 13° to 15° C range. The exceptions were in the US Navy Sea Lab and the Helgoland, a German habitat. The idea was to place the habitat offshore from the Marine Science Center with the full support of the Marine Science Center. This has been lacking in previous scientific habitats. Our concept involved the full support of a sophisticated station and we felt that would produce much more effective work.

The main advantage of saturation diving for people who need a great deal of bottom time, much more than scuba can afford, is that they only have to decompress once. Another advantage is that if the habitat was at 24 m one has an unlimited excursion time from 24m to 45m. The diver can come back to the habitat with no decompression. If one goes below 45m there is some decompression to pay. This allows much more in-water time. If the habitat is in 24m of water, one can work upwards to about half of that, say to about 12m and downwards to 45m for an unlimited time. In a word the purpose of this project was to enable the marine scientists to do very long bottom times.

There are advantages with a habitat when compared to a surface saturation which is what is used in the commercial diving industry for deep diving operations. They saturate on the surface in a chamber and transport the divers down to the work site in a bell. After the dive they bring them home to the chamber on deck. That kind of diving is generally mixed gas (helium and oxygen) and very deep and it is the only way they can operate. In the scientific arena the habitat is in relatively shallow water and the advantage is that the people inside can enter and leave the habitat at will. They do not have to be transported by a bell, which requires a crew to move it. It is a significant advantage. We have had some scientists enter and leave the habitat up to 12 times a day. If one had six of them out of the bell and two or three of them are on different schedules there is quite a problem if you are saturated on the surface. A habitat does have definite advantages.

One example of what we wanted to study was an algae which only grows below 24 to 30m and it has been totally unstudied because with scuba you simply cannot get enough work time at those depths to accomplish anything worthwhile. The plants may grow 15m or more long with blades sometimes two metres wide. The algae settles along the bottom with the current. Underneath the fronds is the offshore fish nursery where all the little baby fish grow up. Studying this would have a tremendous impact on fisheries research, yet it was totally inaccessible to scuba divers. Our concept was to put the habitat within a very short distance off-shore and run an umbilical to it for support. It would open up tremendous areas for study.

In 1980 we contracted for a conceptual design. Then for detailed engineering and then another contract for its construction which was done in Texas over the past year. It was completed about May 1986. The habitat is 12m long, 2.7m in diameter. It has three compartments, a wet porch, a work area and a main living area (Figure 4). It is not much different from Sea Lab or many of the other habitats. As a physiologist it was quite fascinating getting involved in the engineering of this. There are so many implications on the physiology and the medical aspect going through the engineering. The concept involved making it



Figure 4. A cutaway view of the George Bond Habitat. From the left it shows the wet porch, the ablutions area, work space for scientists, living and sleeping areas.

positively buoyant with a heavy base plate and raising and lowering it through a launching system so it can be moved, floated to another site or just at the surface for restocking and what have you.

There were a number of medical problems we encountered in the development phase. Excursion tables from saturation were not in existence. There were a set of excursion tables in the NOAA manual but not for the depths and the gases we wanted to use. Repetitive excursion tables were simply non-existent. If scientists were to go out repeatedly during the day, go down and then back to the habitat, they needed repetitive dive tables. This was contracted to Dr Bill Hamilton in New York. He has developed a set of repetitive excursion tables for saturation diving and he is still working on that problem. That was the first and immediate problem we had because you could not do the excursion diving we were planning without such tables. We decided to use air for excursion diving and normotoxic nitrox for the habitat environment. The nitrox composition varies with depth as far as exact percentages go. Below 15m one cannot use air for saturation because of oxygen toxicity to the lungs. Reducing the oxygen percentage creates additional problems from the higher proportion of nitrogen. One has to be very careful over decompression and narcosis.

The tables are now in existence although they have not been tested. NOAA is trying to decide how to deal with that problem. As with all new tables how do you determine that they are valid? The last I heard they were proposing to test them on people. That is a very difficult proposition and I do not know whether that will happen or not.

Of equal concern was chronic thermal stress. With the water temperatures we have in temperate areas that is a serious problem. The saturation times we were expecting were 7-10 days per mission. We were looking at anywhere from 4 to 8 hours in the water per

day. The acute thermal stress is something that most people immediately think of but that was less concern than what we call chronic thermal stress, the progressive loss of heat over a long period of time. This leads to a loss of performance, which was the primary concern. UMS held a workshop in June 1985 on the subject. The workshop did not solve any problems, it merely emphasized that the problem existed. Our proposal to solve this was to use hot water to heat the divers. That meant they had to be tethered divers. A wet suit or a dry suit, passive insulation, might be suitable for very short duration dives. But the purpose of the habitat was to do long duration dives. The only feasible method of diver heating that we could find was the hot water approach. Commercial diving uses hot water systems extremely effectively. So we planned to install full hot water capability to all the divers. We also designed in a hot tub as full body immersion is the most effective rewarming method there is. We had to rename it diver rewarming something or other because you simply cannot talk to bureaucrats about hot tubs in habitats and have them take you seriously, even though it is an effective means of accomplishing rewarming.

The third aspect was nitrogen narcosis. A normoxic nitrox at 36m is the equivalent of air at a depth of 47m. 36m was our deepest expected depth for the habitat, therefore we would be at a saturation or storage depth where the nitrogen partial pressure was the equivalent of air at 47m with excursions deeper than that. That brings a significant performance detriment from narcosis. There was another UMS workshop on narcosis and saturation diving. It is a very difficult problem. The workshop did not solve the problem. There were many suggestions, but the fact remains that the diver is breathing that partial pressure of nitrogen and there is no easy way around it. Since then some people have suggested that one can train oneself to increase ones performance under the same narcotic conditions. They are still trying to prove that

point. I do not know that they will. This factor is probably of most concern because the whole purpose of the habitat was to put people on the bottom to work efficiently and effectively as Marine Scientists. If they have the narcosis level you would anticipate 47m, how valuable is the data that they are gathering? After they send their papers in to a journal will the editor send it back asking what effect does the level of narcosis have on the accuracy of your data. That is a very serious question for people who might use the system.

The other medical aspect is treatment procedures. How would you treat during saturation or on the surface. We have gone through a variety of scenarios and came up with a long list of answers. For example, if one is saturated at 36m and a diver on an excursion surfaces with an air embolism, should he be taken back down to the habitat or to the shore chamber? Does another diver come up to get him? That would mean that there were two individuals with a guaranteed problem. We have been over a whole series of possibilities. We decided that there would always be someone in a boat available on the surface and if someone did surface they would pick them up and take them to the chamber on the barge, rather than take him back down. Under no circumstances was anyone allowed to surface to help that individual.

Medical standards for divers in saturation are supposed to be somewhat higher than surface diving standards, but the scientists are the same so we went through item by item and decided whether the differences were necessary. We tried to eliminate the usual nuisance of ear and skin problems associated with saturation diving by installing a combined heating and dehumidifying system which permitted a shirt sleeve environment. Humidity and temperature can be controlled very well nowadays. It was not possible 20 years ago in the older habitats but now it is quite possible and not that expensive.

We also needed a PTC, a personnel transfer capsule, because the depth was going to be significantly greater than 15m. If one operates in less than 15m they say the diver has about 20 minutes, if he accidentally surfaces, to get into a chamber and be recompressed. I do not know how true that is, they have done it two or three times at Hydrolab but with any great depth you have zero time. So any transfer to the surface has to be in a pressurized vessel. But there were not enough funds to purchase a new PTC so I got the diving company Oceaneering International to donate a PTC, usually referred to as a bell, which we refurbished totally last year. Also, we were given a deck decompression chamber that the PTC mates to which was going to be placed on the support barge, and we refurbished that. Oceaneering also donated a control van to run both chamber and PTC. NOAA paid for all the refurbishing.

The George Bond is currently in St Croix, Virgin Islands. Why is it there as it was built for Catalina? The reason is very simple. The Hydrolab habitat had failed its hydro test. The Hydrolab habitat is probably the most famous scientific habitat of the early 1970s. A lot of productive work was done from it in the Virgin Islands. Hydrolab was originally in Freeport, Bahamas and then it was transferred to St Croix, Virgin Islands. NOAA operated it almost continuously from about 1972 until 1985. Well over 1,000 scientists have saturated in Hydrolab, a great deal of good work was done from it. It is operated with air, saturated at less than 15m. It is very spartan, and very inexpensive to operate. Last year it could not pass its hydro test so it has been sent to the Smithsonian Museum where it is currently on exhibit.

NOAA obtained the eight year old habitat from Hawaii, the only other habitat in the US, to send to St Croix about 6 months ago. It was going to replace the Hydrolab habitat. Then it failed its hydro test and they had to scrap it. NOAA only had one left, the one we were building. Politically NOAA requires a habitat in St Croix, therefore they sent the Catalina habitat to St Croix at the very last minute. So unfortunately our project is no longer in existence. There have been 65 habitats built in the last 20 years and there is only one left. All the other habitats have either gone to the scrap pile or some other means of disposal. The George Bond is the only habitat that is currently in existence that is operable. Well it is not really operable yet, but very close to it. They currently do not know how they are going to operate it in St Croix and at best it will probably be two years before it goes in the water, if ever.

WAYSTATION PROJECT

However something much more productive for the marine science community came out of this project. We developed what we call a waystation (Figure 2). This is again an old concept, the open bell, which has been used for many years for decompression in commercial diving. However we modified it somewhat so that it is positively buoyant attached to the bottom, rather than negatively buoyant attached to the ship. Its prime purpose is to provide a dry area for decompression, but more than that it is a refuge, it is a work station. It can be raised from any depth without surface effect and is essentially self contained in many ways.

We built the waystation or bell or refuge and operated it for two years while the habitat was being constructed. We operated it quite successfully and to me it is a very useful technique for not only scientific work but perhaps some other kind of work, excavation of sites and that sort of thing, where one needs very long bottom times. The combination of the waystation and tethered diving is a program that we ran for a few years. The first thing we had to do was train the marine scientists in tethered diving because in the whole United States there were only two who had had any training in tethered diving. Everybody else uses scuba exclusively. So we set up a training program. We built the waystation on a shoe string budget. NOAA was very happy to provide the money for building a habitat but they absolutely refused to fund something as low tech as the waystation. Although this proved in the end, at least Catalina Island, to be a much more practical, cost effective tool. A habitat is very very worthwhile if you have firstly a lot of money to operate it and secondly a large contingent of scientists who have to work very long bottom times at that particular site.

We quickly learned that the transition from scuba to surface air required a little finesse, a standard commercial oronasal mask simply did not fit well on a 95 lb young lady going to sit on the bottom and count fish. We found it much more appealing to use simpler systems. We bought a cheap mask and installed the communications and a regulator into it. It made the transition from scuba very easy and it worked well. The communications worked well. I think each mask



DIVING CAPABILITIES AND EQUIPMENT

TETHERED OR SCUBA DIVING

EXTENDED BOTTOM TIMES WITH O, DECOMPRESSION DRY ATMOSPHERE FOR DECOMPRESSION

LIMITED HOT WATER CAPABILITIES FOR WARMING DIVER'S WET SUIT

LOW PRESSURE AIR FOR TETHERED DIVING

HIGH PRESSURE AIR FOR FILLING SCUBA BOTTLES WITHIN THE WAYSTATION

PNEUMOFATHOMETER FOR ACCURATE DEPTH READINGS

HIGH INTENSITY U/W LIGHTING FOR NIGHT RESEARCH FULLCOMMUNCIATIONS: DIVER TO DIVER, DIVERS TO SURFACE, WAYSTATION TO SURFACE

THE ABILITY TO SEND VERBAL DATA TO SURFACE FOR TAPE RECORDING

Figure 5. Diagram of the National Undersea Research (NUR) Program and USC waystation.

cost us \$40.00. We bought 50 of them in a box and at a very low price, probably \$20 apiece. When the mask perished we simply threw it away and put the regulator and communication into another one. Compared to a Kirby Morgan type mask, which runs around \$1200 each, this was a very inexpensive way to operate. It may seem hard to believe that communication in this mask was as good as in other types of full head gear, but they were very close. Once the individual learnt how to enunciate and speak properly they worked quite well and they fit just about everybody.

We have communication, hot water, low pressure, high pressure air, oxygen BIBS (built in breathing system) for decompression in the waystation. There are two seats the divers can climb on inside and be completely out of the water. They can write up their notes while they decompress, they can talk to each other and to the surface. They are held down by a clump weight on the bottom. They are suspended from the bottom up, that is they are floating. The divers can crank the waystation down or up. We would set it down the bottom and then when the divers were ready for decompression they would raise it to their first decompression stop and go on oxygen.

There are a couple of comments I would like to make in closing on this waystation and tethered diving approach, I think it is something that might be applicable in a number of situations. The productivity that came out of it was incredible. The main advantages were unlimited air and hot water. We ended up diving the scientists five to six hours a day at depths that ranged anywhere from 15m to 36m. For all of that 5 or 6 hours they were highly productive. The tether supplied them with air, communications and hot water and the hot water was the thing that made the difference. When they got back to shore they were not fatigued at all. They went right to their labs and worked for another six hours. That was the number one observation that came out of this. That they continued to be productive after a six hour dive. I do not know how many in this room have ever done a six hour dive in 15° water. It is very taxing on the system. Yet by using hot water we essentially eliminated the stress.

The bell can be launched when we want to use it. It floats until the air is pumped out of it. We tow it with a small boat to the site then dive down and attached it to the clump weight and then winch it down to a stable position at whatever depth we are working. There are two tethers on the waystation, so two divers can work independently 60m away from the waystation (Figure 5).

We have a Navy surplus boat that we converted to a dive boat. We put a compressor and the diving manifold and the communication system on it. The standby diver sits on it. As we could not fit the hot water system into the boat we built a little raft for it. The heating system has a diesel boiler and pumps hot salt water down the hoses to the diver. One can regulate the temperature of course depending on the length of the hose. We can move the whole system to a new site in about two hours and do another dive. I feel that this bell and tether technique was the most productive thing that came out of the six year project for building an underwater habitat.

Somebody asked whether there is a future for saturation diving. I am having a hard time deciding whether it is a dinosaur or 20 years ahead of its time. I think in the commercial diving field saturation diving is on the way out. However in some of the other fields it might be 10-20 years away, or maybe it is a dinosaur. As I said earlier, in 20 years of development nothing much has changed. The techniques were there 20 years ago, and yet after 65 habitats there is only one operational one, so that should tell us something I suppose.

Dr Andrew A Pilmanis, PhD, who was one of the guest speakers at the SPUMS 1986 Annual Scientific Meeting, is the Associate Director of the University of Southern California Catalina Marine Science Center, Santa Catalina Island, California.

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TREATMENT AND RESULTS OF

THIRTY HYPERBARIC CASES AT THE

RECOMPRESSION FACILITY HMAS STIRLING

Andrew Robertson

The therapeutic recompression facility at HMAS STIRLING in Western Australia was opened in November 1984. Since that time it has treated nearly forty diving and hyperbaric cases from the Western Australian area. This article is a review of the first thirty cases treated there.

BACKGROUND OF PATIENTS

In this survey, we have looked at the factors of age, sex, diving experience, training and military status. (See Tables 1 to 5)

<u>TABLE 1</u>

AGE OF PATIENTS

AGE	DIVING CASES	Carbon Monoxide (CO) Poisoning
10-20	1 (3.25%)	1 (50%)
20-30	12 (43%)	0
30-40	12 (43%)	1 (50%)
40-50	2 (7.5%)	0
> 50	1 (3.25%)	0

TABLE 2

	<u>SEX</u>	
SEX	DIVING CASES	CO POISONING
Male	23 (82%)	1 (50%)
Female	5(18%)	I (50%)

TABLE 3

DIVING EXPERIENCE

NUMBER	%
4	141
5	18%
10	36%
8	28.5%
1	3.5%
	NUMBER 4 5 10 8 1

<u>TABLE 4</u>

DIVE TRAINING

TRAINING	NUMBER	%
Self-taught	8	29%
Dive Training	18	64%
- Commercial	6	21%
- Dive Clubs	2	7%
- Unknown	10	36%
Professional	2	7%

TABLE 5

<u>STA</u>	<u>FUS OF DIVERS</u>	
STATUS	NUMBER	%
Military	1	3.5%
Civilian	27	96.5%

PRE-DISPOSITION TO DECOMPRESSION SICKNESS

In this review, we look at the factors of omitted or insufficient decompression stops and of repetitive dives (see Table 6).

Using the Royal Navy Physiological Laboratory Tables as a reference, 88 per cent of the DCS patients either omitted completely or performed insufficient decompression stops. 64 per cent were also repetitive diving. Only 12 per cent actually obeyed the tables.

TABLE 6

PREDISPOSING FACTORS IN DCS CASES

FACTORS	NUMBER	%
Insufficient or No		
Decompression Stops	22	88%
Repetitive Diving	16	64%
Within Diving Tables	3	12%

PRESENTING SYMPTOMS

These symptoms were wide and varied, as expected. The most common symptom seen in the decompression sickness (DCS) cases was joint pain (28 cases). This was notably shoulder and neck pain (15 cases), followed by knee pain (5 cases), and elbow pain (4 cases). Other important symptoms include tingling in the extremities (6 cases), paraesthesia (9 cases) and general symptoms of malaise, which included lightheadedness, headache, lethargy and nausea (21 cases). In cerebral arterial gas embolism (CAGE), the most common symptoms were disorientation and decrease in consciousness, whilst in carbon monoxide (CO) poisoning, the most common symptoms were drowsiness and memory disturbances.

DELAY BETWEEN SYMPTOMS AND TREATMENT

In many cases long distances must be travelled before reaching the chamber. In this survey, we looked at the delay between symptoms and recompression treatment (see Table 7).

<u>TABLE 7</u>

DELAY BETWEEN SYMPTOMS AND TREATMENT

CAGE	DCS	CO	
	Р	OISONING	6
2			
2	-		
-	3	1	
1	15	1	
-	2		
-	4		
-	1		
	CAGE 2 - 1 - - -	CAGE DCS P 2 - 3 1 15 - 2 - 4 - 1	CAGE DCS CO POISONING 2 - - 3 1 1 15 1 - 2 - 4 - 1

DIAGNOSIS

There were 2 cases of CO poisoning, 3 cases of CAGE and 25 cases of DCS. The DCS cases were 6 neurological (3 cerebral, 2 spinal and 1 vestibulo-cochlear), 17 musculo-skeletal (10 with peripheral nervous system involvement and 7 without), 1 respiratory, 1 skin and 1 gastro-intestinal.

TREATMENT

The initial treatment given to each of the patients was reviewed. This is a summary of the chamber treatments only, and not of the first aid or adjuvant therapies (see Tables 8 and 9).

Of the 30 patients treated, there were 19 who relapsed (63 per cent). These included 18 cases of DCS and 1 case of CAGE. These cases were treated with RN Table 61 (75 per cent), RN Table 62 (8 per cent) and 5 Metre Soaks (17 per cent). (See treatment table diagram in the next column).

At discharge, complete resolution was achieved in 24 cases (80 per cent) while six had some residual deficit. This was mainly mild joint pain or paraesthesia in the extremities. These six cases included one delayed treatment of a CAGE case and five cases of DCS with delays of between 4 days and eight weeks.

COMMENTS

The majority of the patients in this series were males between the ages of 20 and 40 years, with a reasonable amount of experience. They had mainly been divetrained but a significant number had been self-taught, and this was especially true amongst the pearl and abalone divers. They were almost entirely civilian divers.

TABLE 8

TREATMENT USED

TABLE	CAGE	DCS	CO POISONING
RN 62	_	13	1
RN 62 E	1	11	-
RN 54	1	-	-
OTHERS	TABLE 6	TABLE 6	15 M O ₂
	(MALDIVES)	(THAILAND)	SOAK

<u>TABLE 9</u>

DELAY, RELAPSE AND RESOLUTION RATES

DISEASE	NO.	DELAY	RELAPSE	RESOLUTION
CAGE	2	< 6 Hours	0	Yes (2)
	1	1-7 Days	1	No (1)
DCS	3	< 24 Hours	2	Yes (3)
	15	1-7 Days	10	Yes (12)
				No (3)
	2	1-2 Weeks	2	Yes (2)
	4	2-4 Weeks	3	Yes (3)
				No (1)
	1	> 4 Weeks	1	No (1)







Adherence to the diving tables was very poor and as there is a risk of decompression sickness even whilst staying within the tables (12 per cent of those this series said that they had dived within the tables), the risks of going outside the tables are obvious. Repetitive diving only helps to exacerbate these risks.

CAGE was treated generally within six hours. The one exception to that was initially treated in a chamber in the Maldives twenty-four hours after the onset of symptoms. DCS, however, presented usually at 2 to 3 days with substantial numbers up to 4 weeks and one at 2 months.

The majority of the cases treated were musculoskeletal decompression sickness (57 per cent), though it is noted that three cases of CAGE (10 per cent) occurred during this period.

Treatment was usually with a RN Table 62 or an extended RN Table 62. Although initial improvement was usually good, 63 per cent relapsed. This was probably due to the delay in commencement of treatment. Of these relapses, 67 per cent resolved completely with further treatments.

This review illustrates the cross-section of cases that could be expected to present at a treatment facility. The question of delay before the patient presents is an important one, regardless of whether it is due to travelling delays or patient reticence at presenting. As it directly affects the chance of relapse and eventual recovery, efforts should be made to educate divers to keep treatment delays to a minimum.

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BEER, BUBBLES AND THE BENDS

THE BIOPHYSICS OF BUBBLE FORMATION IN DECOMPRESSION SICKNESS

HP De Decker

NOTES

It would be useful if the reader could pour himself (herself) a tall glass of slightly chilled beer (for illustrative purposes!).

All pressures are given as atmospheres absolute (ATA). Although not strictly SI, this unit has been used, following its popularity in diving literature, because of the ease of conversion from depth to ATA. Each 10m increment in depth equals an increase in pressure of 1 ATA. For the purists 1 ATA = 1 kg.cm².

I am indebted to Paul Hanekom of the Research Diving Unit, Department of Oceanography, University of Capt Town for the use of his library and for stimulating my interest in diving physiology.

I am fascinated by beer, and not only because of its inebriating effect. To me it illustrates some of the most fundamental aspects of that most dreaded of divers' diseases, the bends. Decompression sickness

(DCS) or the bends, is an illness that follows a decrease in environmental pressure which is sufficient to cause the formation of bubbles from inert gases dissolved in the body tissues. It occurs in pilots and tunnel workers suddenly exposed to a large decrease in pressure. It is mainly seen, however, in divers who return from depths where the increased hydrostatic pressures cause high partial pressures of nitrogen in their tissues. This extra nitrogen is then released as bubbles if the ascent is too rapid. A beer, of course, is far from being a complete model of this complex syndrome with its cascading haematological effects, but it serves a useful purpose in illustrating the most fundamental aspect of DCS, bubble formation. And that is what we will explore. Stare into the depth of your beer to where the bubbles appear as if by magic from a single spot in an unending rising string of pearls. How do they form? What hidden forces shape their burst into existence? How does this relate to the bends?

DECOMPRESSION SICKNESS

It has been known since the middle of the last century that DCS is an illness related to bubbles in the blood and tissues.¹ Before 1968, however, literature on bubble formation was virtually non-existent, mainly due to the difficulty of actually observing bubbles in vivo, and to the use of subjective endpoints such as pain, paralysis and other clinical manifestations.² Until quite recently, no direct observations of bubble formation had been made in vivo and the link between the bends and bubbles could only be inferred from post mortem investigations. This meagre evidence. however, was used to construct decompression tables by which divers currently calculate a safe ascent rate from any depth. Although the tables are usually effective for the prevention of DCS, this is obviously an unsatisfactory basis for its treatment. Effective treatment of the disease necessarily requires detailed knowledge of bubble formation as this is its initiating event. But before explaining how the bubbles form, we need to know what they are. For answers, we look at the bubbles in your beer, and then see if we can apply our knowledge to living tissues.

THE BUBBLES

Physics

Let us first determine the forces which act on a bubble in a liquid or in tissues. For a bubble to exist, the total gaseous pressure inside the bubble must be equal or greater than the crushing pressures exerted on it.³ The crushing pressures are:

- 1. the ambient pressure (Pamb), which by Dalton's Law equals the sum of the constituent gas pressures, ie. Pamb = $PN_2 + PO_2 + PCO_2 + PH_2O$ etc (1)
- 2. the tissue pressure (Ptissue), or the pressure the tissue exerts in resistance to deformation, and
- 3. the pressure due to surface tension (Py) of the bubble surface. This is given by Laplace's law as the relation between surface tension (y) and the radius of the bubble (r): P = 2y/r (2)

It is obvious from this relationship that Py is negligible in large bubbles, but that very small bubbles are subjected to extremely high crushing pressures. For a bubble to exist, therefore, the gaseous pressure inside the bubble (Pbubble) must be equal to or greater than the crushing pressures, ie.

$$Pbubble \ge Pamb + Ptissue + Py$$
(3)

The bubbles in your beer consist of CO_2 gas. While the beer was under pressure, the CO_2 was in supersaturation, but bubbles could not form since Pamb was very high. As you opened the beer, Pamb was reduced and the CO_2 could come out of solution and form bubbles. Similarly, a diver is subjected to high Pamb at depth, but when he (rapidly) ascends, Pamb is reduced and the supersaturated gas (N₂ in this case) can come out of solution and form bubbles. This, however, still does not explain their formation.

If you peer closely into your beer, you will notice that the bubbles only arise from the walls or bottom of the glass. Submerge a solid object into the beer, and bubbles will form on its surface as well. This illustrates an important principle:

- 1. bubbles only arise from solid surfaces, or conversely,
- 2. bubbles do not arise de novo from the liquid.⁴

Does this mean that bubbles never arise de novo? If you shake your beer it will be obvious that they can, indeed, arise de novo. The turbulence in the beer causes sufficient reduction of local ambient pressures (Pamb) to allow the formation of additional gas bubbles.⁴ However, in the still glass of beer, the bubbles only form at tiny cracks of imperfections in the glass. Since the walls of the crack are solid, Ptissue will have no effect, and, as the gas/liquid interface is flat (Figure 1a), surface tension (PY) will be negligible. At gaseous equilibrium, the pressure inside this "nucleus" equals the dissolved gas pressure in the liquid. When any gas inside the liquid is in supersaturation however, like the $\rm CO_2$ in your beer, it will diffuse into the nucleus and expand its volume. The surface of the nucleus will bulge out into the liquid and, of course, its growth will be resisted by the surface tension of the curved surface. If the critical radius (eqn. 2) is exceeded, a bubble will form, break away and float to the surface (Figure 1). This then leaves the nucleus free to generate the next bubble.^{2,3} The process will continue for as long as the gas is in supersaturation and if you stare at your beer for too long, all the super-saturated CO2 will come out of solution, and your beer will be flat.

Now all this may sound like sophisticated bar talk, but how much of it is relevant to the study of the bends?

Detection of bubbles

A major breakthrough in the study of the bends was achieved when methods were developed for the noninvasion detection of bubbles.⁵ The existence of bubbles during DCS were now confirmed and experiments on animals and humans could be performed to determine their sites of origin, intravascular course and fate. Bubbles were first detected by using radiographs, electrical conductance and ultrasound.⁶ The Doppler ultrasound method proved the most successful, but is limited to moving bubbles only. Subsequent methods, using pulse-echo ultrasonics,⁵ or the velocity of ultrasound through tissue⁶ have enabled the detection of static bubbles as well. By

FIGURE 1



The formation of a bubble at a crevice in a solid. Adapted from Reference 4.

using these techniques, it was possible to demonstrate that the minimum stable bubble size in tissues or blood is $10\text{-}20\mu\text{m}\text{.}^5$ A more surprising result was the detection of bubbles in divers who remained well within decompression limits and who showed no symptoms of DCS. These were termed "silent bubbles" and were shown to lead to significant haematological effects.⁸ These effects, however, will not be discussed here.

Site of origin and intravascular course

Surgical procedures have recently also been used to determine the origin of bubbles and their course in the blood. By examining the microvasculature of the hamster cheek pouch, as well as its femoral artery and vein during decompression, Lynch et al⁹ have managed to show that bubbles probably first arise in the venous vessels, and only under certain conditions enter the arterial system. Only during "explosive ascents", when decompression from 7 ATA to 1 ATA occurred rapidly, were bubbles observed in the arterial system.

By using a Doppler probe, Butler and Hills⁷ have shown that the lungs act as excellent filters for microbubbles in the venous system. By placing the probe over the venous drainage of the lungs (precordial position) and infusing bubbles of varying size intravenously, they were able to demonstrate that almost all bubbles larger than 22 μ m were filtered from the blood by the lungs. Hills and Butler¹⁰ repeated the experiment using a Coulter counter to determine the bubble size in blood drawn from the venous sinus of dogs. They could then show that bubbles would pass through the lungs if (1) too many bubbles were produced in the veins (2) vaso-dilation occurred in the lungs, or (3) the lungs were damaged in some way, for example by excessive oxygen exposure. Ohkuda et al¹¹ confirmed this result by showing that no air entered the arterial circulation under normal physiological conditions after intravenous infusion of 2 ml of air per minute into sheep for 3 hours.

Hemmingsen and Hemmingsen¹² had previously shown that bubbles seldom formed intracellularly, even at very high supersaturation levels of nitrogen. Even if bubbles did eventually form, their effect was overshadowed by that of the bubbles in the extracellular fluid. The picture now seems a bit clearer. The site of origin of the bubbles is probably extracellular and usually in the vein or extracellular fluid. Now that we know a little more about them, we can continue to investigate the mechanism of bubble formation.

Bubble formation

The formation of bubbles in decompressed animals and humans is described by two opposing theories.¹³

- 1. the mechanical stress theory,² generally known as "de novo nucleation".
- 2. the micronuclei theory.

Both can occur in your beer under different conditions, but which is more correct in its description of bubble formation in DCS? Let us investigate each of these, and some their variants.

The de novo nucleation theory

This theory predicts that bubbles will form de novo, ie. where none existed before, within the blood or extracellular fluid due to highly localised negative pressures induced by some kind of mechanical stress. The theory is neatly summarised in the equation which describes the conditions for nucleation:²

$$\Delta F = 4\pi r \{ y - rPbln(Pt/Pb) \} / 3$$
(4)

where F is the free energy of nucleation required to create a bubble of radius r,

- y is the surface tension of the bubble,
- Pt is the tissue pressure,
- and Pb is the bubble pressure.

In a nutshell, the theory holds that during the random thermal motion in all liquids, submicroscopic cavities are formed, which collapse immediately under normal physiological conditions due to attractive intermolecular forces. If these cavities are placed under a negative pressure (mechanical supersaturation) of 100-1000 ATA, however, the (mechanical intermolecular forces will be counteracted and the cavities will expand to reach stability.14 Bubble formation can also occur de novo if the liquid is heated, or gas-supersaturated to approximately 150 ATA.¹⁵ Since this pressure is equal to a depth in seawater of 1490m, while the bends can occur from any depth deeper than 10 m,³ this theory seems to be inadequate.

The micronuclei theory

The first experimental evidence for this theory proposed by Harvey et al,¹⁶ was provided by Evans and Walder.¹⁷ Harvey et al¹⁶ had managed to reduce the number of bubbles formed in water on decompression by applying hydrostatic pressure to the water before decompression. Pre-pressurising forced the gas in the micronuclei into solution, thereby reducing the number of nuclei available for bubble formation. The experiments of Evans and Walder¹⁷ used this observation and are so elegant that I will give a brief resume here.

The brilliance of the investigators is shown by their choice of experimental animal: the common shrimp, Cragnon cragnon. There are two obvious advantages in that choice. Firstly, the shrimp is transparent, so that any macroscopic bubbles formed can readily be seen. Secondly, the shrimp is found at all depths in the sea and is consequently not affected by large changes in hydrostatic pressure. If the shrimp contained micronuclei, and their number could be reduced by pre-pressurisation, a degree of protection from bubble formation should be attained. Evans and Walder¹⁷ decompressed 100 shrimps, of which 50 had been pre-treated by pressurisation to 400 ATA for 2 minutes. The results of their experiment are shown in Table 1.

TABLE 1

The incidence of bubbles in pressure treated and untreated shrimp, Cragnon cragnon, after decompression (From Reference 17)

It is obvious from these results that the destruction of micronuclei leads to a drastic reduction of bubble formation.

Further evidence for the micronuclei theory came from Vann et al¹³ who pressure treated Wistar rats before subjecting them to decompression. The reasoning was that if bubble formation occurred de novo, the pressure treatment would enhance the bends due to the higher supersaturation level in the rats' tissues. If gas bubbles formed from micronuclei, however, the pressure treatment would decrease the incidence of DCS due to the destruction of micronuclei. Their results showed a decrease of almost 20 per cent in the incidence of DCS after pressurisation and decompression. The second hypothesis was therefore accepted: bubbles form from micronuclei which could be reduced by pre-pressurisation. Now, of course, our next question follows logically: how do the micronuclei form?

The origin of micronuclei

In an extension of their micronuclei experiment Evans and Walder¹⁷ gave a clue to the origin of micronuclei per se (not the bubbles). They repeated their previous experiment with 100 pre-pressurised shrimps, of which 50 were stimulated to perform vigorous flexural contractions and rapid leg movements. The results of this experiment are given in Table 2.

	TABLE 2		
	NO. WITH BUBBLES	NO. WITHOUT BUBBLES	
Pressure treated only	4	46	
Pressure and stimulation	16	34	

The effect of stimulation on bubble formation in pre-pressurised shrimp, Cragnon cragnon, after decompression (From Reference 17)

Bubble formation was obviously increased in the stimulated shrimps, possibly due to the increase of micronuclei in their tissues caused by movement. Evans and Walder¹⁷ attributed the increase in micronuclei to "high impulsive stresses" set up during movements in the tissues of the shrimps. It is certainly a well known fact that vigorous physical activity during ascent from a dive increases the probability of the bends in humans.

Tribonucleation is a theory originally proposed by Hayward¹⁸ and later invoked by Vann and Clark¹⁴ to attempt an explanation of the origin of micronuclei. The theory proposes that large local mechanical supersaturation pressures are generated when two closely opposed surfaces are separated in a liquid. This movement will produce super-saturations directly proportional to the product of the liquid viscosity and the velocity of the separation of the surfaces. Tribonucleation has been used by Unsworth et al¹⁹ to explain the "cracking" of joints as the formation and collapse of cavitation bubbles within the joint capsules. It seems to me, however, that this brings us back full circle to do novo nucleation. In fact, the theory has not been used subsequently to explain the origin of micronuclei, which is still poorly understood.

The role of surfactants

A further development of the micronuclei theory is that nuclei without solid bases (eg. in blood) are surrounded and stabilised by "skins" of surface-active compounds.²⁰ This followed from the reasoning that if these micronuclei were larger than $1\mu m$, they should rise to the surface of the liquid, while if they were smaller than 1µm, they should dissolve due to the intense pressure generated by the surface tension of the gas/liquid interface. Since neither event occurred, the existence of a stabilisation membrane of surfactants of varying permeability was postulated. At gaseous equilibrium, the membranes are permeable, but they become effectively impermeable if the hydrostatic pressure is increased rapidly to sufficiently high levels.²⁰ Very little is known of the surfactants, except that they are composed of non ionic hydrocarbon moieties²¹ and that the desorption of the surfactants from the gas/ liquid interface would presumably lead to the dissolution of the micronuclei.

The Varying-Permeability Model

Recently, Yount and Strauss²² and Yount et al²³ have developed a model from the micronuclei theory to describe a wide range of cavitation phenomena related to decompression. The model, called the varyingpermeability model, has since been used successfully to describe decompression limits (limits to which decompression can be taken before bubbles start to form) in rats and humans²⁴ as well as fingerling salmon.²⁵

The model is a mathematical description of the formation of bubbles of a minimum radius at different supersaturation levels, crushing pressures crumbling compressions (the mechanical strength of the surface membrane) and surface tensions. Ultimately, the model relates the ambient pressure to micronuclear radii, and therefore, by Laplace's Law (eqn. 2), to the number of bubbles that are able to exist at that pressure. Above a certain minimum radius, nuclei originally present will grow to form macroscopic bubbles, while those below the minimum radius will either dissolve or remain as micronuclei.²⁵

CONCLUSION

The varying-permeability model is not a model of the bends, but only of the initiating step in this complex disease. It has already been of practical benefit, however, in the prevention and treatment of DCS. This is an excellent example of how basic research into fundamental principles can lead to advances in practical knowledge.

By now your beer will be flat. I hope, however, that it will not have been wasted, but has served a useful purpose, I am sure that when next you enjoy an ice cold lager, like me, you will not see it as just another glass of bubbly liquid, but will spare a moment to reflect on the bends in your beer. Prost!

SUMMARY

Decompression sickness (DCS) is an illness which follows a decrease in environmental pressures which is sufficient to cause the formation of bubbles from inert gas dissolved in the body tissues.

Bubble formation, therefore, is the initiating event in the pathogenesis of this syndrome with its cascading haematological sequelae.

Effective prevention and treatment of DCS can be derived from a better understanding of bubble formation.

Bubbles exist when the gaseous pressure inside the bubble is equal or greater than the crushing pressures (ambient pressure, tissue pressure and the pressure of surface tension).

Two theories describe bubble formation: the mechanical stress theory (de novo nucleation), and the micronuclei theory. The latter is currently favoured.

The micronuclei theory holds that bubbles form from microscopic gaseous nuclei, either lodged in a crevice, or stabilised by surfactant "skins" of varying permeability. When any gas in the surrounding liquid is in supersaturation, it will diffuse into the nuclei, expanding its volume until a bubble forms.

The varying-permeability model was developed from the micronuclei theory to quantitatively describe cavitation phenomena. It has been successful in predicting decompression limits for various animals and man, illustrating the usefulness of theoretical models to practical medicine.

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A NEW SYSTEM OF GIVING OXYGEN TO DIVERS IN AN EMERGENCY

Ken Wishaw

I would like to describe a simple alternative method of delivering high concentration oxygen to awake patients with decompression sickness. I believe it is an improvement on systems at present in use, and does not appear to have been described before.

With the increasing emphasis on the value of normobaric oxygen therapy as soon as possible after the onset of decompression sickness, this method should be of interest to members of SPUMS and to divers generally.

The following are the desirable features of such a system:

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It should:

- 1) Supply a high concentration of oxygen,
- 2) Be economical in oxygen usage as oxygen supplies on boats or during transfer may be limited,
- 3) Comfortable to use,
- 4) Easy to assemble and fit to patient,
- 5) Cheap,
- 6) Not susceptible to corrosion,
- 7) Easy to clean,
- 8) Compact,
- 9) Able to be used for ventilation of non-breathing patient with oxygen and still be able to ventilate if the oxygen supply is exhausted.

The use of the Mapleson C style resuscitation bag attached to an anaesthetic mask and held in place by a Clausen harness has been popular.

However its disadvantages are:

- 1) Effective sealing of the mask on the face is difficult, leading to air entrainment,
- 2) It is uncomfortable,
- 3) Oxygen requirements are high if rebreathing is to be prevented,
- 4) It cannot be used for mechanical ventilation if oxygen supplies are exhausted.

More recently there has been marketed an adaptor (Bendeez) which allows scuba regulator to be attached to oxygen cylinders.

This has the advantage of using a familiar and comfortable delivery system, namely the rubber scuba mouthpiece. A noseclip, or a first aider's fingers pinching the nose or wearing a diving mask, is required to prevent breathing through the nose diluting the oxygen with air. The Bendeez system delivers 100% oxygen and is economical with the use of oxygen.

The disadvantages of this system are its inability to be used as a ventilator and the potential hazard of fire if incorrect lubricants have been used in the regulator.

For completeness, portable recompression chambers should be mentioned but they are beyond the scope or pocket of most divers.

The system I have devised meets all of the above desirable features.

A resuscitation bag is used, but instead of an anaesthetic mask and harness, a standard rubber scuba mouthpiece is fitted. Most brands of scuba mouthpiece fit the 22 mm male taper of resuscitation valves. This simple conversion eliminates all the disadvantages described above. A nose clip is required or the patient can wear his diving mask to seal the nose or a first aider can pinch the nose shut.



Figure 1 Laerdal bag with Scuba mouthpiece fitted to non-rebreathing bag in place of a face mask.



<u>Figure 2</u> Laerdal bag system in use showing, from the bottom, collapsible oxygen reservoir bag, air entrainment valve, self-inflating bag, non-rebreathing valve and nose clip.

The Laerdal bag consists of a Laerdal non-rebreathing valve and a self-inflating bag with an air entrainment valve and a collapsible oxygen reservoir bag. This brand of resuscitation bag is probably the most popular brand in New South Wales and is the standard bag used by the NSW Ambulance Service.

The advantage of this particular brand is the valve itself, which is of low resistance, non-corrosive and can be used both for mechanical ventilation (resuscitation) and spontaneous respiration. Most similar valves available will not allow spontaneous respiration. Being a self-inflating bag, the system may be used for ventilation and will continue to do so if the oxygen supply is exhausted.

Gas analyses of the Mapleson C and Laerdal bag systems were performed using a mechanical lung and chest model to simulate a 70 kg patient. With a tidal volume of 700 ml and respiratory rate of 10 breaths per minute. Various fresh gas flows were used.

When the fresh gas flow was equal to the inspired minute volume, namely 7 litres per minute, the inspired oxygen concentration was over 97% in both cases. However it must be remembered that the Mapleson C system is a rebreathing system and "acceptable" levels of rebreathing require a fresh gas flow of at least twice the minute volume. The Laerdal system is more economical of oxygen. The fresh gas requirement for the Laerdal system can be judged clinically by ensuring the reservoir bag is fully inflated at end expiration. (Significant air entrainment only occurred at fresh gas flows of less than minute ventilation. Taping over the air entrainment valve is thus not necessary).

The Laerdel resuscitation equipment (with whom I have no affiliation!) is readily available through the medical supply company, Drager and can be purchased in a compact, durable, plastic box complete with Guedal airways and masks but without a nose clip or scuba mouthpiece for just over \$200. (Laerdal Silicone Resuscitation in Compact Case, catalogue number 87 00 03). To give oxygen an oxygen regulator (reducing valve) and a flow meter will be needed.

This combination of Laerdal bag, scuba mouthpiece and nose clip represents a simple and significant improvement for oxygen delivery to the awake patient.

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FUTURE MEETINGS

IX INTERNATIONAL CONGRESS OF HYPERBARIC MEDICINE Hilton Hotel, Sydney, 1-4 March 1987

UNDERSEA AND HYPERBARIC MEDICAL SOCIETY

Hyatt Regency Hotel, Baltimore, 27 - 30 May 1987

Peter Horne

The increasingly popular pastime of recreational scuba diving is today becoming better understood by both the diving community and the general population. However, there are three aspects of the underwater world which still tend to dominate the headlines and affect the "average Joe's" perception of our activity, SHARKS (giant man-eating fish a la Jaws); THE BENDS (an agonising and usually fatal disease which afflicts all divers who surface too quickly, according to midday TV soap operas); and RAPTURES OF THE DEEP, a form of drunkeness caused by diving "too deep", invariably resulting in drowning when divers hand their scuba gear to passing fish!

Fortunately, those of us who actually dive are very well educated in recognising such hazards and know how to deal with them ... DON'T WE?! We all know that you carry a big knife to fight off sharks, and that responsible divers who follow the decompression tables <u>can't</u> get Bent, only fools who break the rules will cop it! (Don't worry DES, I'm only kidding!!)

The third well-known problem, <u>nitrogen narcosis</u> ("Raptures" to the ignorant peasants) is a bit trickier to handle. Divers going to depths of around 30 metres or more enter the realm of the esoteric and infamous "Narks", and funny things begin to happen.

In the warm, clear ocean waters of tropical regions, divers often report feeling elated at depth, enjoying the experience immensely. Likewise, people put under pressure in recompression chambers frequently have a great time, giggling and marvelling at their "Donald Duck" voices in the warm, secure confines of However, such reactions seem to the chamber. disappear totally when you substitute this relatively comfortable environment (in which you are dry, breathing normally and able to communicate clearly with others) with the cold, dark and often silty world of the waterfilled caves and sinkholes. Instead of euphoria, divers may experience negative and potentially very dangerous effects which need to be quickly recognised and carefully handled, not the easiest of things to do when you are heavily taskloaded.

The waterfilled sinkholes of the Mount Gambier region of South Australia are of special significance to me, and whilst they rarely provide me with cause to feel euphoric at depth, such experiences are nevertheless possible ... especially if you happen to see the stars shining through a mirror-calm surface at midnight in Piccaninnie Ponds from a depth of 33 metres! Although many sinkholes may contain crystal-clear water at depth, the limitations of underwater torches are such that the clarity generally cannot be fully appreciated and it is very much like doing deep ocean night-diving in many cases. Seeing stars or clouds from such depths underwater though may inspire feelings of genuine awe, and might in fact be little influenced by narcosis, who can tell?

During the past few years, I have undertaken nearly 200 cave dives to depths of 30 metres or more, and have encountered narcosis frequently enough to almost regard it as an unwanted dive buddy who is up to no good. Strangely, many of my cave diving companions



even today still seem to have an ego problem when it comes to admitting to ever being narked, and many in fact still believe that the effect can be totally conquered by simple concentration. Like seasickness, they think that it is "all in the mind". The often-published statement that narcosis only affects those of us with low intelligence makes it hard for me to understand why some real "dead-heads" I know never get affected ... perhaps they are simply too stupid to know that they are narked! The relatively simple act of merely swimming around, shining a torch on the wall does not really give narcosis a good chance to express itself, unless something goes wrong. However, because I have frequently been involved in doing some sort of manipulative or other skill such as taking bearings, reading thermometers and writing notes, I have been acutely aware of how nitrogen narcosis can affect even the simplest of operations. The danger with concentrating on overcoming one aspect of narcosis is "task fixation", which can result in divers losing track of other important things like monitoring air supplies, whilst putting their total concentration on the "problem" at hand.

My first awareness of narcosis usually occurs around 30m, where I feel a little light-headed and find my vision becomes a little out of focus (my built-in corrective lenses might explain this a bit). At this stage I tend to recheck my torches, the reel, and all other gauges to be sure that all systems are "go" before descending further. Between around 35 and 50m, narcotic effects are much more noticeable, my fingers feel rather numb and writing on a slate requires a bit more concentration than usual. Opening containers with floating lids becomes a tricky operation, often resulting in the glass bit dropping away into the gloom as the lid shoots off towards the surface. Sudden feelings of warmth and unusual comfort sometimes cause immediate feelings of concern, until that disgusting thing many divers do to keep warm! Strange timewarps occur here, with the actual bottom time slipping away very quickly although getting your buddy to respond to a simple signal seems to take 3 to 4 times as long. My every movement is calculated and very slow.

Bevond a depth of 50m. I notice more serious effects. especially in confined regions where silt lies waiting to be disturbed by a single careless movement and may add to any feelings of anxiety. The air often takes on a metallic-oily taste which makes me feel a little nauseous (who serviced my regulator last time?) and feels cold and thicker, almost liquidy. Exhaled bubbles tinkle like glass shards hanging above an open window, and I can stare at the larger dome-shaped ones for ages as they rise out of view without bursting, unlike those released in shallower waters. Over 60m, I believe that divers are exhibiting suicidal tendencies as they may suddenly find themselves in a trance-like psychological state which I call "DREAMTIME", an effect which causes you to lose your normal awareness of reality, making the whole dive seem to be strangely unreal and almost dreamlike in nature. This is a very scary feeling, because although you somehow know its not really a dream, you realise that you can't seem to get back to "reality" and must force yourself to go through the actions of great importance such as checking your air, time and all the rest.

Dreaming may be fine if all goes well, but if a problem, ANY problem, should come along, a "Dreamtime" dive can suddenly become a very dramatic, short-lived nightmare. This is always a very real danger of deep diving, our old mate "Murphy", is particularly active at such depths.

Although people seem to adapt reasonably well after repeated exposure to depth, I suspect that narcosis does not go away. Once any unexpected feelings (such as light-headedness or slightly blurred vision) are accepted by the subconscious, anxiety is naturally diminished and divers can more readily handle the other problems that narcosis generates. The only way that this can occur is through frequent repeated exposure to such depths.

One of the most worrying aspects of severe narcosis is the loss of peripheral awareness divers experience. A diver may snag his foot in a line or his guideline might stop reeling out, but it can often take many seconds for him to realise that this has happened. In a truly critical situation, such slowness of thought could mean the difference between life and death.

An example of how people react under pressure is related here from my own experience. During a dive when I was the leader and at a depth of around 55m, I was just starting to carefully enter a rather confined silt-laden region when I became aware of the hurried flashing of my buddy's torch, as he swept it around from behind me to gain my attention. Normally in seadiving or shallow-water conditions I would have turned around immediately, assuming something was wrong. However, on this occasion I became extremely agitated, thinking such things as "What the hell's that guy playing at? Can't he see I'm concentrating on entering this nasty little hole? He probably only wants to show me something unimportant anyway ..." However, I then realised where we were and, realising that he might have become entangled in the line or had an air problem, I proceeded to turn around. Fortunately in this instance, it turned out that he was worried about his camera imploding, as he had forgotten to leave it in shallower water and didn't want to take it any further. The interesting thing is that it took probably 10 to 15 seconds for me to tealisa that there could have been a problem, even though my buddy's torchflashing had immediately gained my attention. Experience is the teacher!

It is well known that nitrogen narcosis affects people differently and can affect an individual in different ways on separate dives as well. I have been surprised to find myself almost narcosis-free after awakening in a fatigued condition, and conversely, I have been seriously affected at shallower depths than usual even though I was looking forward to the dive and was wellrested. Narcosis may also attack at unexpected times, on one occasion I was free of the effect until we turned around and started out, some 10 minutes into the dive, when it suddenly clobbered me like a ton of bricks! I also suspect that there are residual effects. One reason for this belief is the very relaxed, "switched off" feelings I have noted upon returning towards the surface, and promptly spending several minutes of valuable decompression time at the wrong depth!

Nitrogen narcosis is the biggest killer of cave divers in this country, with lack of safety lines to cave entrances being the major factor in the other cases to date. Unfortunately, the beauty and clear water found in many sinkholes can only start to be appreciated at depths of 30m or more, and the unavailability of improved deep-diving equipment such as the expensive Heliox mixtures etc. means that today's deep-divers, like our pioneers, must still carefully plan and execute deep dives, being forever on the lookout for narcosis. Such diving, I believe, is something akin to rockclimbing, not everyone can (or should) do it. It is, after all else has been said, a hostile environment we are going into and is thus potentially dangerous. It is a strangely beautiful world down there, totally unlike anything else in our everyday experience, with the potential for great discovery lying in the silt of every cave.

Hopefully, nitrogen narcosis will one day soon shed its aura of mystery to inquisitive researchers, so that we may explore the unchartered depths with calm hands and clear minds!

EDITORIAL COMMENT

Sinkhole diving in South Australia has been controlled by the CDAA on behalf of the South Australian Government for over 10 years with an excellent safety record, except for two divers who disregarded CDAA regulations and dived in a sinkhole they were not qualified to dive.

Peter Horne's article is of great importance, emphasising a diver's eye view of nitrogen narcosis and its effects.

It is obvious from the article above that diving to depths below 30 m is dangerous. The diver may accept the risk, make a mistake and die which then puts those who have to search for the body at a similar but unnecessary risk. US Navy figures¹ show that few diving accidents occur shallower than 15 m, 0.69 per thousand dives. From 15 to 30 m the rate is more than tripled to 2.32 per thousand dives. Between 30 and 61 m that rate is more than doubled to 5.4 per thousand dives, which is nearly eight times the accident rate for dives to less than 15 m.

The Editor wishes to draw readers' attention to Regulation 61 of the Cave Divers' Association of Australia (CDAA) published in August, 1986.

Regulation 61 reads

No CDAA member shall dive deeper than 37 m in a categorised sinkhole.

(Penalty: Suspension of membership for a period determined by the Committee).

Unfortunately the CDAA AGM in September 1986, while voting to accept the regulatiom as published, decided that: "The '37 Metre Rule' (Rule number 61) has been declared to be a recommendation and not a Rule. A formal amendment to the Regulations will eventually be issued", 1uoted from the synopsis of the minutes published in the November 1986 issue of the CDAA publication "Guidelines").

REFERENCE

1. Blood C and Hoiberg A. Analysis of variables underlying US Navy diving Accidents. Undersea Biomed Res. 1985; 12.3: 351-360.

Peter Horne's address is 3/9 Muriel Avenue, Somerton Park SA 5044.

THE DIVETECH RECOMPRESSION CHAMBER

The prototype of a 2-man transportable recompression chamber (RCC) will soon arrive in Australia to be set up at Port Lincoln Hospital, following trials at The Royal Adelaide Hospital. Designed primarily for the stabilisation and transport under pressure of a victim and an attendant, the chamber offers these unique features.



- 1. It is lightweight and transportable. It can be moved using a 1 tonne flat-bed truck, an Iroquois helicopter, a Beechcraft King Air, or lifted by 4 men.
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- 3. It has full gas monitoring equipment for the inside environment.
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The recompression chamber is one part of a twochamber system. The second or transfer chamber locks onto the portable recompression chamber, or can stand alone as a decompression "working tool" while the emergency evacuation chamber is in use. Together they form a multilock multiplace recompression system for the treatment of decompression sickness and pulmonary barotrauma, allowing the transfer under pressure of all ancilliary services.

A spares and maintenance service will be provided to purchasers, along with technical and operator manuals.

For further details contact:

Mr Bob Sands Managing Director Divetech 15 Gladstone Street KEMPSEY NSW 2440 Australia

Telephone: 61 65 628010 Fax: 61 65 628090 "Divetech"

LAW REPORT

INQUEST FOR FACTS OR INQUISITION FOR HERESY? A CAUTIONARY TALE FOR ALL WHO DIVE

Douglas Walker

THE DIVE

The basic facts are simple and not in dispute. There were seven divers who met for a dive, five being trained and guite experienced (two were awaiting formal notification of status as assistant instructors) while two were newly qualified and making their second post-course scuba dive (they had not yet received a certification card issued by the Instructor group whose manual, program, and logo were used for the course which they had just completed). The divers knew each other and the victim's first postcourse dive was with one of those taking part in this dive. The dive platform, a rock shelf, was only a little above sea level and was covered a few inches deep by water whenever the half metre swell hit the rocks. It was covered by sea squirts (tunicates) and the divers decided it would be safer to walk on it without fins, which would be put on after making their water entry. The sea was calm and this appeared to be a highly suitable entry point for their dive.

It was a warm day so two of the group cooled off first by making a short snorkel swim, then got back onto the rocks and donned their scuba gear. The two notices got kitted up while in the parking area above the rocky point, checking their own and each other's equipment before descending to the platform to join the others. This check disclosed that the victim had mistakenly attached his regulator to his tank upside down so that the hose came over his left shoulder, an error which was again noticed (and corrected) when they joined the others and were checked by one of the experienced divers who had chosen to complete his preparations closer to the water.

To identify the divers and their actions in this incident the following code has been used:

- DA "Assistant instructor" training and had taken a Rescue Course
- DB "Assistant Instructor" training and had Resuscitation and Rescue training
- DC Trained and experienced
- DD Trained to Advanced Diver level
- DE Trained with one year experience
- DF Novice making 2nd scuba dive since recent course
- DV Victim, novice, 2nd scuba dive since recent course

First into the water were DA and DD. They turned. floating quietly in the calm water 5-6 metres off the rocks, to watch the others prepare to enter the water to join them. The DE, who was standing with DV near the edge of the platform ready to enter the water (without fins) shouted out a warning that a larger wave was approaching unseen by DA and DD. The swell was hardly noticed by the two divers in the water but washed over the rock platform a few inches deep and caused both DE and DV to lose their balance. They were helped up by DF and DB, who had been on higher rocks preparing their equipment. DE then entered the water and swam out to DA and DD before donning fins. However DV was less fortunate, another wave now arriving and washing him once more across the rocks and into a gulley. With the help of DB and DF he

succeeded in putting on his fins while in this gulley during the respites between the next two water surges. He was weighted down by his tank and backpack and water was draining into the gulley off the rock platform after each wave so his easiest and most safe option seemed to be to swim down the gulley until he reached the quiet waters off the rocks. He did not show any signs of panic and part inflated his vest when so requested by DF, who joined him. DF, who wore a wet suit and buoyancy vest but not his scuba, told DV to start using his regulator, which he did, and held onto DV's equipment as they traversed the turbulent waters close to the rocks. While this was occurring DB was on the rocks donning his scuba. He swam out and joined DV and DF at the critical time when DV was starting to panic, making rapid arm and leg movements, breathing in a shallow and rapid manner, and grasping his companions and dragging them down.

They were now fairly close to where the other divers were waiting, which was the easiest exit option, so DB signalled to them for assistance. They rapidly joined him and then it was noted that DV was unconscious, his demand valve was no longer in his mouth (when replaced it was spat out again) and there was froth coming from his mouth. The part inflated buoyancyvest was keeping him at the surface so they did not drop his weight belt, believing that it helped to keep him vertical in the water. As he was unconscious and frothing from the mouth they decided to get him out of the water rapidly rather than attempting in-water resuscitation, and they were close to the exit point at this time. He was pulled up onto the rocks and turned on his side while DB quickly removed his own backpack before commencing to give resuscitation. The beach inspector from the nearby beach arrived a short time later and noticed that head extension was incomplete. He had an "Oxy Viva" and attempted resuscitation without success. The problems caused by regurgitation of fluid into the victim's mouth were noted by him also.

THE INQUEST

At the inquest counsel were present to represent not only the widow and the instructor who ran the training course but also three of the divers, with a solicitor appeared on behalf of another diver. It was the widow's counsel who was largely responsible for the course of this inquest, searching for any evidence of culpable mistakes by anyone involved in the dive or the training course. As a result the basis of questioning was entirely on what the Instruction Manual stated and there was no effective examination of whether, in the circumstances of this dive, the actions taken were reasonable. Not one iota of thanks was offered to any of the divers for their valiant attempts to assist the victim. The coroner noted that he was not bound to observe the usual rules of evidence, a point not commonly known. Also appearing was a representative from the instructor organisation and two persons there to put forward the interests of two government departments.

The instructor organisation noted that the course wrongly promised that those who successfully completed it would then receive a certification card carrying the name of the diving organisation whose instruction manual was used on the course and whose logo was displayed in the dive shop. Because of an unresolved question of conforming to the organisation's list of course requirements the instructor had been notified that he held a non-teaching status. The witness carefully made no comment on the course the victim had taken beyond noting the apparent omission of pool dives (however there had been five open water dives). Evidence was given that the end of course written test was unsupervised and the pupils had easy access to their note books at this time, but no evidence was offered on whether this compromised the diving ability of pupils who completed the course. It is noteworthy that DF, the second of the novice divers on this dive, acted in a highly commendable manner and had obviously reached a high standard of ability.

In contrast the input from governmental sources seemed to be designed to support proposals to introduce laws to control diving instruction rather than to have any relevance to the unfortunate incident. It was claimed that the introduction a few years previously of a voluntary code for dive shops hire of equipment and supply of air "had dramatically reduced for some eighteen months to two years" diver deaths but over the past twelve months the numbers showed a rise again, which was taken to mean that voluntary methods were failing. There was no evidence offered to support this conclusion, one which the Provisional Reports on Diving Related Fatalities certainly in no way suggests. Neither was there any evidence offered that the dive course inadequately prepared the victim. The other government department's expert was equally unhelpful to this investigation because he made no attempt to relate the facts of the case to his comments. He correctly noted the absence of a dive flag, which was a nil factor in the incident, made a suggestion that the victim may have become fatigued while he tried to orally inflate his vest (which was inflated by hose supply from his full tank), and misrepresented evidence about the water conditions. The beach inspector stated the sea was calm but about every 30-40 minutes there would be a group of larger waves. As the divers observed the sea over 20 minutes before commencing their dive, there was a pre-dive check made of the novice divers, two of the experienced divers waited in the water a little off the entry ledge watching carefully as the others prepared to enter the water to join them in guite shallow calm water, the dive management showed care was taken even though the dive was loosely structured.

Criticism was levelled by counsel on the divers because a series of actions differed from those given as being correct in the instructor organisation's book. This assumed that all advice given in the book was beyond dispute as the only safe and correct action permissible. Such was not established. It was assumed that climbing out of a gulley filled with rather turbulent water was the only safe course of action, to seek a respite in calm water was incorrect. It was assumed that one must release the weight belt of the victim even should he be adequately supported by his buoyancy vest, and the assumption was made it was necessarily always better, even should a victim be unconscious, frothing from the mouth and a suitable safe exit area be close by, to attempt in-water resuscitation by EAR. There is no documentary proof to support such views. The actual degree of support afforded the diver by his vest, an important detail, was never ascertained.

CONCLUSIONS

The fatality appears to have been truly "a misadventure" and the result of an inexperienced

diver being unexpectedly, and several times, tumbled about by waves coming out of a sea which appeared to be calm, these sweeping across the rocks on which he was standing. Though he managed to don his fins, and followed advice to inflate his vest and use his regulator, he was less in control of self management than was apparent and apparently removed his demand valve while being assisted out to the calm water, this being associated with an increasingly uncontrolled breathing pattern and rapid but ill coordinated limb movements. He then inhaled water and rapidly became unconscious. The rescue and resuscitation response by his dive companions was unsuccessful. There was no probing of whether the divers were correct in their decision to take the victim to deeper calmer water, to leave the weight belt in position, not to attempt inwater EAR (two had taken resuscitation and life saving courses but were not asked whether these courses adequately prepared them for the situation they encountered). Although great play was made of the instructor's membership-status in the instructor organisation, his poor written notes recording the training of his pupils, the absence of any real supervision of the end-of-course written test, the omission of pool dives in favour of (protected) open water dives, and the misrepresentation to pupils that they would be granted a card of certification by the instructor organisation, nothing was found to link this fatality with any course deficiency.

Another coroner (case 170) has stated "that in activities where accidents can and do occur, the question must always be asked: How well instructed or prepared was the person for the particular activity in which he was engaged?" Here the evidence points clearly to the victim being trained and also having the correct equipment in good working order. It is to be hoped that coronial investigations continue to follow the guidelines defined by a New Zealand coroner who said that it was not the coroner's function to establish blame, it was his function to establish the identity of the deceased and when, where and how he died. Counsel have different objectives, the interests of their client being their sole consideration.

DIVING SAFETY MEMORANDA

Department of Energy Diving Inspectorate Millbank London SW1P 4QJ

DIVING SAFETY MEMORANDUM NO. 3/1986 DETERMINATION OF OIL VAPOUR IN COMPRESSED AIR

It has come to our notice that Draeger gas analysis tubes of the type 10/a and 1/a can underestimate the quantity of oil vapour in a gas sample. Draeger are currently publishing new calibration cards for these tube types. It is recommended that the finest droplet size (0.3 to 0.5mm) calibration is used for assessing oil in breathing gas.

Diving companies are to ensure that a valid calibration card is used if they are utilising Draeger tubes to verify compressed air to BS 4001 part 1.

The revised calibration cards can be obtained from:

Draeger Safety PO Box 4 Blyth Northumberland NE2X 1HA Thames Hospital Board Mackay Street Thames NEW ZEALAND

18 December 1985

HIATUS HERNIA AND DIVING

Dear Sir

I was recently asked to assess the cardiac status of a 67 year old experienced diver who on 2 or 3 occasions in the middle of this year, experienced attacks of breathlessness lasting for 10 to 12 minutes. On two occasions he became alarmingly breathless when surfacing from 100 feet and on another occasion when descending to 10 feet.

A stress ECG was conclusively negative. Eight and a half minutes of exercise on a bicycle ergometer using a modified Bruce Protocol was tolerated well. However, a chest x-ray showed an incarcerated hiatus hernia with a large fundal gas bubble to the left of the heart. He then disclosed a dyspeptic history of 35 years with increasing dyspepsia, with regurgitation and retrosternal pain for 5 years. He tends to vomit at dinner time and is breathless on putting on his shoes.

Past and recent investigations have indicated incarceration of the hiatus hernia from 1981. Inconstant motility disorders of the oesophagus were demonstrated by Mr JF Carter of Green Lane Hospital in August 1983. Current films indicate that approximately the upper half of the stomach is lying in the chest cavity and is of para-oesophageal type with the cardia lying just below the level of the left side of the diaphragm. Of interest too, is that an IgG Monoclonal band of 1795 (normal range 800 - 1800) is present. This is of unknown significance as there is no evidence of myelomalignancy, such as Bence Jones proteinuria, elevated calcium, excess of plasma cells in the bone marrow etc. The haemoglobin is low at 13 g/1. His other laboratory tests have proved non-contributory.

In my opinion, the gas bubble in the incarcerated hernia in this patient could contract at depth and reexpand at the surface causing discomfort and distress. However, this hypothesis does not answer the question of why his symptoms have only developed recently in the presence of a 35 year history unless his low grade anaemia and paraproteinaemia are of importance. It also raised the issue of hiatus hernias and diving in general. Should a hiatus hernia be a contra-indication to diving?

Yours faithfully

SA Maar Physician

Reprinted by kind permission of the Editor, NEW ZEALAND JOURNAL OF SPORTS MEDICINE.

The Editor South Pacific Underwater Medicine Society

Dear Sir

In view of the importance generally attached to the wise use of Australia's natural resources, the Australian Committee for the International Union for Conservation of Nature and Natural Resources (ACIUCN) has developed a policy and guidelines for the establishment and management of marine and estuarine protected areas (MEPAs). This will be presented to State and Commonwealth agencies to help them develop better strategies for managing both coastal and offshore natural resources and environments.

The Committee has developed a package of materials which is aimed at improving public awareness of the need to protect our marine heritage and to provide for resource and habitat maintenance in Australian waters. Part of this package is an article which identifies the needs and the role that MEPAs can play in meeting them. A copy of the article is enclosed for possible use in your newsletter or for distribution to your members.

Diving is generally compatible with the objectives of MEPAs and will be allowed to continue in most areas. Any thought by divers that MEPAs are contrary to their interests is unwarranted.

Under the ACIUCN policy, protected areas are established in full consultation with user groups. Divers are in a unique position to assist in the task of ensuring that the range and quality of recreational opportunities on and under the water are maintained. Your members' input to proposals for protected areas is valued and needed.

Increased understanding of and co-operation in conservation programs is essential if present and future generations of Australians are to experience and enjoy the long term benefits of marine and estuarine environments, Help from your organisation is needed to encourage governments to adopt the ACIUCN policy, and to understand the need to conserve, protect and use wisely Australia's nearshore and offshore marine areas.

If you would like further information about marine and estuarine protected areas, and/or a copy of the Policy and supporting papers, please write to

Secretary Marine Reserve Sub-Committee of ACIUCN GPO Box 636 Canberra ACT 2601

Yours sincerely

Pam Eiser President Australian Committee for the International Union for Conservation of Nature and Natural Resources

Phone: (02) 211 5366

4 June 1986

THE ZIERING ALPHABET

Bob King

Underwater communications can be a problem. Apart from the standard signals which are taught as part of every scuba dive course, many divers and Instructors have often developed their own sets of signals. For instance, there's the diver who has decided after the umpteenth time that he's never going to get that doff and don exercise mastered to the Instructor's satisfaction, and in frustration, he signals ...



The Instructor, however, feels that his (or her) professional reputation is at stake, and being an exemplar of Patience and Perseverance (what FAUI Instructor isn't??), decides to give it just one more try. The Instructor then, most politely, but firmly, signals back ...



These signals are unmistakable and deliver the message. Other forms of body language are also used underwater. There will still come the time, though, when you wish to deliver a comprehensive message, but haven't the means with which to do it. Use of the Ziering alphabet can help you give your student the drum without having to surface for verbal discourse.

The Ziering alphabet was first published in the American magazine, "Sport Diver" back in 1980. It was developed by Lezly Ziering, a diver and a modern jazz dance instructor. The truly marvellous thing about it is that it can be learned within 5 minutes. <u>No foolin'</u>!! Have a look at the symbols opposite, then give them a go. You'll surprise yourself with just how many you remember at the very first attempt.

One of the first things to remember is that many of the signs actually resemble the letter you're signing. The signs can be depicted by either the left or the right hand, and <u>even when wearing gloves</u>! What's more, <u>you only need one hand</u> to give each sign.

Here's how Lezly Ziering suggested you form the letter ...

"Straightforward and simple are the first ten signs. A fist with a thumb pointing up is a D to the viewer. For letters E and F, you may use any two or three fingers held sideways. The thumb crooked at the first joint is G. Your palm sweeping parallel to the ground signals the middle line of the H and the small or the index finger points to your I (eye).

"Pretend you're making a gun, then add your little finger for K. Use any two or three fingers pointing down for M and N. The fist with the thumb pointing down is the shape of the lower case p. For Q, insert your thumb between the index and middle finger of your fist. The sign for R resembles the lower case r.

"Into the home stretch: Index finger making a squiggle is S. Take a cup of "T", but don't round the thumb or index finger - that means OK. U, V, W, X and Y are just a matter of mimicking the drawings. Use your arm Egyptian style to form a Z.

"Double letters are made by retaining the letter and pushing your hand toward the viewer twice. Between words, drop your hands or go limp-wristed."

Ziering claims to have taught his students individually in as little as three minutes, with an average learning time of five minutes, The concept is easily grasped by novices, and has a high retention rate.

Norm Bracken suggests, "Each word is ended by a diagonal slashing motion of the hand (none of this limp-wristed stuff!), and a "talking" movement with the fingers is used to signal you are going to start signing, to eliminate confusion with the standard hand signals."

Well Norm, I tried it, and it works! Not only that, but I taught my family the alphabet in 5 minutes flat. It's amazing just how easy it all is. Now I can't wait to try it with my next dive class. I'm going to photocopy the opposite page here and give each of them a copy, and then try it out. I reckon I can just about leave my underwater slate behind. I think I might try to develop a set of numerals too, but that should be easy.

This paper first appeared in FAUI NEWS, 1986; Vol. 8 No. 2, March: 30-31.

Bob King is the Editor of FAUI NEWS. The address of FAUI NEWS is PO Box 1296, INGHAM QLD 4850, Australia.

MYTHS OF DIVING - I

William J Lawrence

The Great Lakes Chapter of the UMS again hosted a panel session on the Myths of Diving during a program on 'Medical Aspects of Diving' at Underwater Canada '85 in Toronto. During the session Sunday 31 March, some 70 sport divers, diving instructors and physicians listened to a distinguished group of diving experts

















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explore various long-standing myths. The panel, chaired by Bill Lawrence, consisted of Dave Henderson, Bill Hamilton, Shane Willms and Sandy Jones.

Dave Henderson exploded three of his favourite myths: 1) the most significant diving problem is "bends"; 2) serious diving problems develop in deep water; and 3) religious adherence to the USN Diving Tables guarantees no bends.

Bill Hamilton chose to focus on two additional myths: the old rewarming theory of hands and feet out of water, and the premise that female divers tolerate cold better than male divers. Bill also recalled that until recently the USN Diving Tables were regarded as the diving bible, He suggested that a revision was in order and asked the audience to consider the USN diving tables as the 'Old Testament' and the newlyreleased DCIEM tables as the 'New Testament'.

Shane Willms made a strong case for destroying the myth that "drinking and drugs don't affect my diving". He cited numerous case histories and references that alert the diver to the contraindications that exist for many over-the-counter preparations as well as for doctor-prescribed meditations. Finally, Shane advised, "If you drive - don't drink."

Sandy Jones presented evidence to dispel the myth that "men are better divers than women". She noted that the only obstacle that confronts female divers is the male ego. Sandy referred to the low incidence of scuba-related accidents among females and the work capacity of female commercial divers who devise alternate schemes for handling strenuous jobs. Finally, Sandy asked that female divers not be restricted to such scuba-related jobs as dive instructor, travel agent, diving shop operator and underwater rescuer. She believes that women have a far greater potential in 'the waterbeds of our planet'.

A lengthy discussion followed the brief presentations made by the panelists. Several areas of interest emerged as topics of very real concern to divers. These are listed below so as to inspire other diving program committees to address these issues. It became very clear from the lively discussion period that sports divers need and want medical expertise. Among the issues that concern divers are:

- the use of antihistamines, antihypertensives and antibiotics
- diabetes and diving
- childhood asthma and diving as an adult
- the use of oxygen following a diving accident
- the status of oxygen as a drug
- pregnancy/menstruation/contraceptives and diving
- vulnerability of females to decompression sickness
- free flow versus demand oxygen delivery
- the incidence of dysbaric osteonecrosis among sport divers
- what constitutes a long bone survey
- the use of contact lenses by divers

The DCIEM 1983 Air Diving Tables were the final focus of discussion. These relatively "bends-free" tables captured the interest of a large number of the audience. Ron Nishi, who was instrumental in developing and testing the tables, explained their use and the testing procedures that guided their development. Given the warm reception afforded the myth panelists and the genuine interest shown in topics related to sport diving medicine, this GLC-sponsored panel has emerged as a highlight of the underwater Canada program. It is hoped that the myths panel, which now has a two-year track record, will become a regular feature of Underwater Canada.

Reprinted, by kind permission of the Editor, from PRESSURE, the Newsletter of the Undersea Medical Society, June 1985: 7.

The address of the Undersea Medical Society is 9650 Rockville Pike, Bethesda, Maryland 20814 USA

PAIN AFTER DIVING

Roy Lewisson

A diver completed a surface oxygen decompression dive (90 feet, 120 minutes) and immediately reported low back pain, which radiated downwards. He felt it was muscular in nature and checked out fine neurologically. There were no unusual characteristics of the dive.

I wanted to run a test of pressure (TOP), however, the supervisor pointed out that it was the company's policy not to allow a repetitive dive within six hours of a Surface Decompression on Oxygen (Sur.D.O₂) exposure! The supervisor argued that since the TOP would require recompression this would be considered a repetitive dive, following a 30 minute delay.

Finally, "when in doubt - treat" so we put him on a USN Table 6. Upon reaching 60 feet there was very, very slight relief and his condition remained stable throughout the treatment. This, of course tied up chambers and a technician for half the shift and no doubt produced a degree of pulmonary oxygen toxicity. All of which may have been unnecessary.

Upon surfacing and for another 24 hours symptoms remained unchanged, then began to resolve without further treatment.

COMMENTS

Clearly, the diver deserves to have his symptoms investigated. The greatest advantage of the TOP is to get him into the chamber, due to the ever persistent "element of denial" that surrounds many cases of suspected decompression sickness. No repetitive diving following a Sur.D.O₂ exposure is one thing, but the rule should not apply to TOP as it is therapeutic compression. Remember, the TOP results in further inert gas uptake, and it could be argued that the hyperbaric oxygen exposure is not a repetitive dive at all. This incident appeared to be a misinterpretation of guidelines, and hopefully, has already been resolved internally. Editor, TRIAGE.

Reprinted by kind permission of the Editor from TRIAGE, The Newsletter of the National Association of Diver Medical Technicians, No. 9, Janurary 1985.

The address of the National Association of Diver Medical Technicians is C/- College of Oceaneering, 272 South Fries Avenue, Wilmington, California 90744, USA.

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ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT COURSE PROGRAMME 1986-1987

Date	Course	Duration (Days)
2 - 6 February 1987	Divers - Advanced first aid	5
9-13 March 1987	Medical Practitioners - Basic UHM	5
16-20 March 1987	Medical Practitioners - Advanced UHM	5
23-27 March 1987	Nurses - RCC attendants	5
22-26 June 1987	Nurses - RCC attendants	5
6-10 July 1987	Divers - Advanced first aid	5

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT COURSE FOR NURSES - RCC ATTENDANTS

Objectives

To provide the technical training necessary for nurses and appropriate para-medical personnel to operate as attendants to patients in a recompression chamber.

Candidates

The course is designed for registered nurses with critical-care training, but other registered nurses, enrolled nurses, and paramedical personnel will be accepted. Candidates must be fit for hyperbaric exposure in accordance with Australian Standard AS 2299 Appendix 1. Each course will be limited to 15 candidates.

Duration

Five days

Course Composition

The mornings will be devoted to theory teaching, with practical sessions each afternoon. Both will emphasize intensive care procedures within a recompression chamber. A detailed course programme is available on request.

Cost

\$A150 enrolment

People interested in this course should either write to:

Mr C Holmes Hyperbaric Medicine Unit Dept Anaesthesia and Intensive Care Royal Adelaide Hospital North Terrace ADELAIDE SA 5000

or ring him on (08) 224-5121

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT COURSE FOR DIVERS -ADVANCED FIRST AID

Objectives

To enable divers to recognise dysbaric illness, to apply appropriate first aid, and to initiate hyperbaric retrieval. The course will qualify candidates to the appropriate regulations and standards.

Candidates

The course is designed for professional divers, but is suitable for advanced recreational divers.

Duration

Five days

Course Composition

The mornings will be devoted to theory teaching, with practical sessions each afternoon. The practical sessions will include an orientation to transportable recompression chambers. A detailed course programme is available on request.

Cost

\$A150 enrolment

People interested in this course should either write to:

Mr J Houston Hyperbaric Medicine Unit Dept Anaesthesia and Intensive Care Royal Adelaide Hospital North Terrace ADELAIDE SA 5000

or ring him on (08) 224-5514

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT COURSE FOR MEDICAL PRACTITIONERS

Objectives

<u>Basic</u>

To introduce medical practitioners to the underlying principles of Undersea and Hyperbaric Medicine (UHM), and to enable medical practitioners to adequately assess the fitness of candidates for either diving or hyperbaric exposure (eg. compressed air work).

Advanced

To enable medical practitioners to provide appropriate initial management of patients requiring hyperbaric therapy.

Both courses will qualify candidates to the appropriate regulations and standards.

Duration

Basic: five days Advanced: five days

Course Composition

The course will comprise both theoretical and practical sessions. A detailed course programme is available on request.

Cost

Basic: \$A250 Advanced: \$A250

Enrolment

People interested in this course should either write to:

Dr D Gorman Hyperbaric Medicine Unit Dept Anaesthesia and Intensive Care Royal Adelaide Hospital North Terrace ADELAIDE SA 5000

or ring him on (08) 224-5116

AVAILABILITY OF THE SAFETY SAUSAGE

As anyone who has looked for a diver at sea knows, a diver on the surface is difficult to spot.

The Safety Sausage (SPUMS J. 1986; 16(2): 59), is a red plastic tube which can be inflated by the divers regulator. It can either be held vertically in the water to indicate the diver's position to a boat or allowed to lie on the surface to aid recognition by aircraft.

There is at present no Australian distributor of Safety Sausages. However the manufacturers, TL Begg and Sons Ltd, PO Box 5216, Moray Place, Dunedin, New Zealand, are willing to supply them wholesale to Dive shops and they are available for members of SPUMS through RJ Knight Pty Ltd, 80 Wellington Parade, East Melbourne VIC 3002 at \$7.00 including postage.

FUTURE MEETINGS

IX INTERNATIONAL CONGRESS OF HYPERBARIC MEDICINE

Will be held at the Hilton Hotel, Sydney from 1 to 4 March 1987.

A draft programme and registration details are available from

Medical Convention Services PO Box 125 Heidelberg VIC 3084 Australia Telephone (03) 458 1333 Telex AA 37803

1987 JOINT UNDERSEA AND HYPERBARIC MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING AND THE TWELFTH ANNUAL CONFERENCE ON CLINICAL APPLICATION OF HYPERBARIC OXYGEN

27 - 30 MAY 1987 The Hyatt Regency Hotel, Baltimore, Maryland, USA

For further details write air-mail to

1987 Joint UMS/HBO Meeting Attn: Jane Artigliere C/- Undersea and Hyperbaric Medical Society 9650 Rockville Pike, Bethesda, MD 20814 USA

SPUMS 1987 ANNUAL SCIENTIFIC MEETING

To be held at the Mendana Hotel HONIARA, SOLOMON ISLANDS 2 to 7 June 1987

Guest speaker Dr Tom Shields, President of the European Underwater Medicine Society.

This is a call for papers which should be sent to the Convenor of the Scientific Meeting

Dr David Davies Secretary of SPUMS Suite 6 Killowen House St Anne's Hospital Ellesmere Road Mount Lawley WA 6050

For travel details contact

Allways Tours 168 High Street Ashburton VIC 3147 Australia