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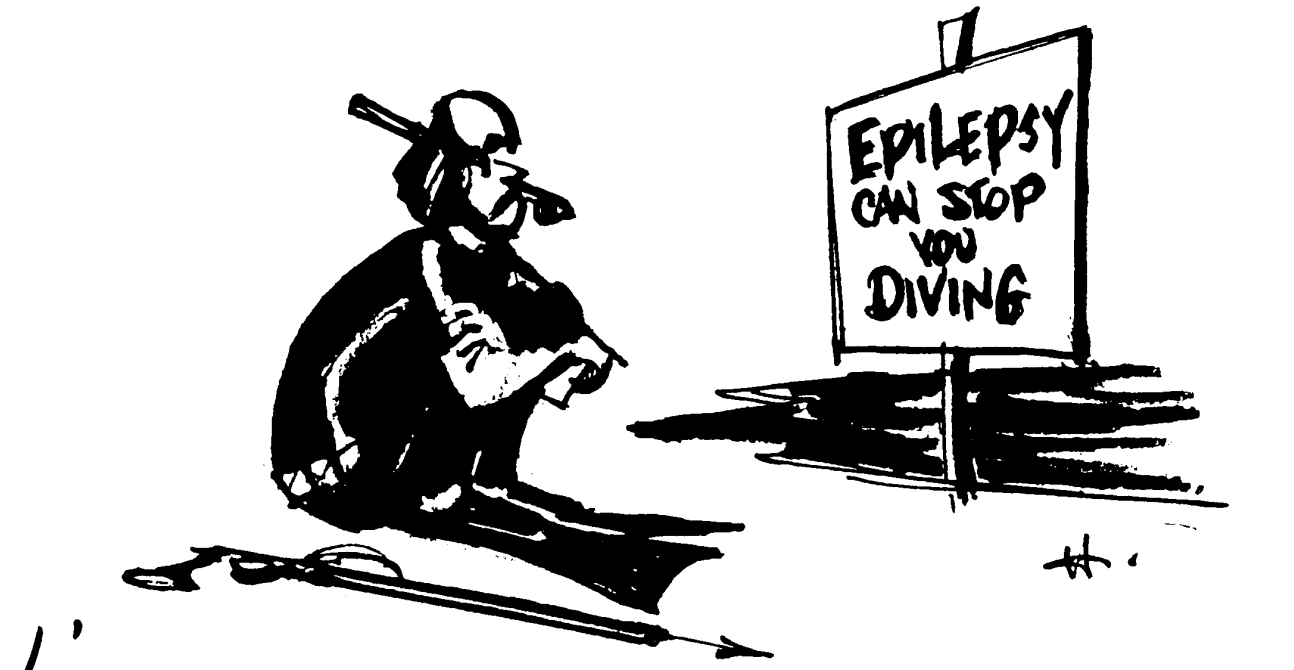

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President	Dr Chris Lourey	25 Hastings Road, Frankston VIC 3199
Secretary	Dr Chris Acott	Rockhampton Base Hospital Rockhampton QLD 4700
Treasurer	Dr John Doncaster	222 Noble Street Newtown VIC 3220
Editor	Dr Douglas Walker	Box 120 Narrabeen NSW 2101
Committee	Dr David Davies Dr John Knight Dr Janene Mannerheim	Perth Melbourne Melbourne
Hon Cartoonist	Mr Peter Harrigan	

EDITORIAL

A chance, non diving, reader might glance at the listed contributions to this issue and conclude that divers are indeed a strange lot to enter an environment which seems to contain so many drastic disincentives. It might be thought that anyone lucky enough to escape the spines of a stone fish, the bite of a blue-ringed octopus or the tentacles of a box jellyfish then has the choice between decompression sickness and cerebral arterial gas embolism (CAGE). A more educated reader might even note that one can get symptoms suggestive of both decompression sickness and CAGE from the same dive. However, in practice, few will ever need to translate into action the information available in these pages on such types of morbidity. To be even more precise, it is the hope of the contributors that their presentations will lead to a reduction in the incidence of the very matters which form the subject of their papers.

The problems of decompression sickness are discussed from the viewpoints of the theory of case presentation and management. It is worth debate at some future time to consider whether the fortunate discovery of a practical way to reduce the risk of decompression sickness among the Hard Hat divers of that time by Haldane and his colleagues would have been of even greater benefit without the seductive gift of a theory which appeared to explain the practical findings. Many tribulations later "the experts" are becoming more humble and flexible in their claims to understand the workings of Nature. Unfortunately, this newfound awareness has yet to diffuse through the ranks of sport divers, who still tend to act as if The Tables were rules that Nature rather than themselves must follow. The Tables remain the most sure defence against decompression sickness, apart from keeping out of the sea (and the sky!), but should be applied with caution. It must be recognised that we do not understand the factors which condemn one person to Spinal Bends while his buddy remains clinically unaffected. Caution indicates the sense of adding safety factors of "extra" depth and time when fatigue, cold, being a woman or having a repeat dive, etc. are involved. Not only are no two people the same but the same person may have different responses to the same 'bubble lead" on different occasions, and nobody has calculated tables to take account of the bubbles which are present at the commencement of all except the first dive. Three or more dives a day may be fun but they certainly enter physiological Terra Incognita, which may be one factor in the seeming excess of cases of DCS now occurring.

CAGE, the acronym to save time and space when talking about cerebral arterial gas embolism, may well be one of those conditions which are underdiagnosed because of the belief that air/gas embolism is a dramatic, and usually fatal, event immediately on surfacing. The review by Des Gorman, and the case reports, should increase awareness and understanding of the condition, leading to a greater willingness to consider the possibility of it having occurred. Immediate post-surfacing neurological symptoms, even of short and self-limiting duration, should be regarded as indicative of the possible presence of gas embolism and managed accordingly.

In dealing with such "respectable" diving medicine conditions there is a risk of forgetting the humbler problems which can affect the safety and indeed the life of a diver. Such conditions include the significance of the accepted potentially adverse factors of an asthma history, epilepsy, diabetes and heart disease. As it is unlikely that any acceptable control will ever become possible of breath-hold divers, and equally certain that some will always evade the medical check-points and become scuba divers despite their adverse health history, it is hoped that every reader who becomes aware of such persons will assist an increase in knowledge by arranging for the CONFIDENTIAL recording of the diving experience of such persons through the reporting system of Project Stickybeak. They must be doing something reasonably correctly to escape inclusion it in the fatality reports!

MINUTES OF THE ANNUAL GENERAL MEETING
23 June 1983

held at the Fijian Resort Hotel, Fiji.

MINUTES OF THE 1982 GENERAL MEETING
Dr C Lourey moved that the minutes of the 1982 meeting be accepted, seconded by Dr Davies. Carried.

TREASURER'S REPORT
Presented by the treasurer, Dr J Doncaster. G Leslie moved that the treasurer's report be accepted, seconded by D Carson. Carried.

CHANGE OF AUDITOR
Dr Doncaster suggested that SPUMS change auditor to Mr Les Newman of Krampel & Newman of Footscray. The motion was proposed, seconded and carried.

NEXT ANNUAL SCIENTIFIC MEETING
To be held at Phuket, Bangkok and Hong Kong, on 8th - 20th April 1984. Guest speaker would be Surgeon Captain Ramsay Pearson, Royal Navy.

ELECTION OF OFFICE BEARERS
The following had been elected by postal ballot and were declared elected.

PRESIDENT	Dr C Lourey
SECRETARY	Dr C Acott
TREASURER	Dr J Doncaster
EDITOR	Dr D Walker
COMMITTEE	Dr J Knight, Dr J Mannerheim, Dr D Davies

There being no other business, the meeting was closed.

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 OF SPUMS

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.

At present the subscription for Full Members is $A30.00 and for Associate Members is $A25.00 Membership entitles attendance at the Annual Scientific Conferences and receipt of the Journal.

Anyone interested in joining SPUMS should write to:
Dr Chris Acott,
Secretary of SPUMS,
Rockhampton Base Hospital,
Rockhampton QLD 4700.

NOTES TO CORRESPONDENTS AND AUTHORS

Please type all correspondence, in double spacing and only on one side of the paper, and be certain to give your name and address even though they may not be for publication.

Authors are requested to be considerate of the limited facilities for the redrawing of tables, graphs or illustrations and should provide these in a presentation suitable for photoreduction direct. Books, journals, notices for symposia etc., will be given consideration for notice in this journal

REPRINTING OF ARTICLES

Permission to reprint original articles will be granted by the Editor, provided that an acknowledgement to the *SPUMS Journal* is printed with the article. Papers that have been reprinted from another (stated) source require direct application to the original publisher for permission to publish, this being the condition for publication in the *SPUMS Journal*.

MINUTES OF THE EXECUTIVE MEETING
29 October 1983

held at the Leichardt Hotel, Rockhampton.

ATTENDING C Lourey, C Acott, J Knight, J Mannerheim, D Davies.

APOLOGIES D Walker, J Doncaster.

Minutes of the last meeting held on 27th August, were read and accepted as correct.

BUSINESS FROM THE MINUTES

1. Indonesian Conference, 1985.

   A reply from the Indonesian Navy still had not arrived. The committee recommended that an alternative venue be considered for the 1985 conference.

2. Thailand Conference, April 1984.

   There still had not been any replies from either the Thai or Singapore Navies about their support or attendance at the conference.

   The committee decided

(i) abandon the Bangkok venue for the meeting, and stay in Phuket until 16th April and then fly to Hong Kong.

(ii) send an invitation to the Thai Navy to attend the Phuket meeting, and not rely on them to participate at the conference.

3. Several complaints had been received about the cost and timing of the conference in 1984.

Action: Ask Allways Travel for a breakdown on expenses etc for the conference.

CORRESPONDENCE

1. Letter of thanks from Prof B Hills.

2. Letter from the National Library of Australia.

3. Letter from the Underwater Explorers Club of WA requesting permission to reprint articles from the SPUMS Journal.

   Action: Permission was granted provided they acknowledged that the article was from the SPUMS Journal.

4. Letter from Dr I Unsworth of the Hyperbaric Unit at Prince Henry's Hospital, Little Bay, NSW.

   He notified the Committee that the next course in Hyperbaric Medicine would be conducted at his unit on 2 - 4 March 1984. A note would be put in the SPUMS Journal notifying members of the course.

CORRESPONDENCE TO THE PRESIDENT

1. Newsletter from the Australian College of Occupational Medicine.

2. Newsletter from the Australian Resuscitation Council.

3. Letter from the Margaret Geer Media Group re the Directory of Australian Associations.

4. Letter from the Commonwealth Dept of Health (Vic Division), concerning the recompression facilities in Victoria. A reply had been sent.

5. Letter from the University of Washington, USA., concerning a conference on the Management of Acute and Chronic Pain, to be held at Truk Lagoon on 25 March - 1 April 1984.

6. Letter from Dr I Unsworth enquiring whether, with the introduction of Medicare, diving medicals would qualify for a rebate?

   Action: A reply will be sent informing him that it will STILL be ILLEGAL TO ITEMISE ACCOUNTS FOR THESE MEDICALS.

7. Letter from Dr Finlay-Jones. He had various complaints concerning the recent Fiji conference (snorkelling arrangements in particular), the lack of compensation to Dr Finlayson when he had to return from the Madang conference early, and the timing and expense of the Phuket conference.

   Action: The President, Chris Lourey, will reply to his complaints.

TREASURER'S REPORT

This was delivered by the President in the absence of the Treasurer. There were no financial problems.

GENERAL BUSINESS

1. Letter from Dr D Walker. The National Association of Diving Technicians of USA had applied for membership.

2. Letter from Dr G Phillips. He queried the use of oral fluids in the treatment of Decompression Sickness.

   Action: A reply will be sent with the appropriate references.

3. Heron Island would be investigated re the suitability for the 1985 SPUMS conference. The secretary will report his findings at a future Executive meeting.

The meeting was closed at 1830 hours.

SECRETARY'S REPORT JUNE 1983 - JUNE 1984

NEW MEMBERS Full 31 Associate 36

ANNUAL SCIENTIFIC MEETING

The Annual Scientific Meeting (ASM) was held in Phuket and Hong Kong in April 1984. The Guest Speaker was Surgeon Capt RR Pearson RN. The academic programme was interesting and stimulating, however, the general overall attendance at the meetings was POOR. This was not only embarrassing for the guest speaker but also for the committee members present. This seems a difficult problem to overcome and regrettably it seems to occur every year.

The diving programme was good and the snorkelling was much better than other years. Let us hope it continues.

At Hong Kong, Dr P Preston (of the University of Hong Kong) delivered a very comprehensive talk on other perils of the South China Sea. These included Japanese B encephalitis, hepatitis, malaria and the odd case of syphilis and gonorrhoea!

Overall I think that the conference was a success. The Annual General Meeting was not held and will not be held until the ASM in 1985.

OTHER MEETINGS

A combined Australian and New Zealand Intensive Care Society (ANZICS) Queensland Branch and SPUMS meeting was held at Rockhampton in October 1983. This was well attended, but not however by SPUMS members. 120 people attended, and only 10 of these were SPUMS members.

NEXT YEAR

Hopefully the ASM and the AGM will be held in April in the Maldives. Two guest speakers have been asked. The scientific programme will be concentrating on other issues besides decompression sickness and gas embolism, and I feel will have a broad range of interests.

I hope to see you all next year.

NEW ZEALAND DIVING-RELATED DEATHS, 1983

Douglas Walker

Four breath-hold and six scuba diving fatalities were identified from the reports of the New Zealand Water Safety Committee. Of the breathhold deaths, two appear to have the characteristics of post-hyperventilation anoxic blackouts leading to drowning, one was an epileptic and the remaining victim was inexperienced, without fins and wearing a borrowed weight belt which was without a quick-release buckle. His buddy was too fatigued to offer assistance to him. Of the scuba fatalities, the critical factors were water power, alcohol, inexperience, and possible cardiac inhibition following aspiration of water while at the surface. Better buddy diving discipline would have reduced the number of deaths.

CASE NOTES

Case BH 83/1

The victim and his buddy were diving for mussels on an offshore reef. Initially the water was neck deep but it became deeper with the incoming tide. The victim was stated to be a good swimmer but this was only the 3rd or 4th time he had "dived". The borrowed weight belt he wore did not have a quick-release. He apparently got into some difficulty and his buddy felt too tired to attempt to offer any assistance. As he could not release his weights, did not wear fins or a buoyancy vest, and was inexperienced in diving, he drowned.
INEXPERIENCE. NO FINS. UNABLE TO DROP WEIGHT BELT. OUT OF HIS DEPTH WATER. BUDDY NON-ASSISTIVE

Case BH 83/2

This was the second time the victim, an epileptic, had gone diving. He was on medication but apparently still suffered fits, usually with a prodromal onset of twitches. He swam to a buoy with his friend, who momentarily lost sight of him while ducking under a rope. Some bubbles were seen ascending but the water was too deep for the buddy to reach the bottom to attempt a search.
INCOMPLETELY CONTROLLED EPILEPSY. SEPARATION.

Case BH 83/3

A group of twelve experienced divers were on a boat-based dive, three pairs being left at a rocky islet. The victim and his buddy scuba dived then returned to sit on the rocks, removing all their equipment including their compensators. Another diver borrowed the victim's mask to enable retrieval of a lost mask. Unfortunately, after the mask was returned a sudden wave struck their position and the mask was washed away, though the rest of the equipment was retained. The victim borrowed his buddy's mask and swam out from the islet to find his property. He called out that he could see it below in about 30 ft deep water, then dived. He failed to resurface and the buddy, maskless, was unable to find his body. It is very probable that this was a post-hyperventilation-blackout (anoxic) death. The weight belt did not have a quick release, but this would be unlikely to effect the outcome of such an occurrence. It is not known whether the scuba tank still contained air.
ALONE. POST HYPERVENTILATION BLACKOUT. LOSS OF MASK LED TO ERROR (SCUBA WOULD HAVE BEEN SAFER). BUDDY UNABLE TO SEARCH.

Case BH 83/4

Separation during spearfishing is common, probably inevitable. This pair was diving in 12 ft deep water and after a separation of only 3-4 minutes the buddy was alarmed to see his friend on the sea bed below him, all equipment in place, lying still. It is likely that this was a post-hyperventilation death despite the medical history of a right middle lobe removal (for bronchiectasis) and moderate-to-severe asthma. He had attended a diving school and was an experienced diver. It is stated that he was a fairly strong willed person who could not be discouraged from diving.
SEPARATION. POST HYPERVENTILATION TYPE DEATH. ADVERSE HEALTH HISTORY.

Case SC 83/1

After a reunion party, which included beer drinking, it was decided to proceed to the nearby river to catch some eels to make a meal. The victim donned his scuba equipment while the others just swam about at the surface. He descended into a hole in the river bed about 15 ft deep and for a time his bubbles were seen. After about an hour his friends became anxious and started a search. Although he had attempted a diving course, and was proud of his certificates, in fact he failed to complete the course and had been told he was not up to standard and needed more instruction. He had, however, continued to scuba dive. There is no record of remaining air being checked, though the tank was fitted with a contents gauge.
ALONE. SHALLOW RIVER. PART TRAINED. PROBABLY OVER CONFIDENT OF ABILITY.

Case SC 83/2

A party of nine divers, all with some experience, were on a chartered boat dive to an offshore island. The boat was anchored and the divers entered the water as buddy groups, the victim and his buddy being the last to enter. It was arranged that the buddy was to catch the crayfish and the victim to hold the bag. The boat's skipper saw the buddy suddenly surface and signal the need for help. He described being tossed about by a sudden surge which tore off his fins and had seen the victim rushed past him by the water flow. The body was recovered later with all the equipment intact. There is no record of anyone anticipating this problem or of it effecting any other members of the group.
WATER POWER NEAR ROCKS.

Case SC 83/3

During a family outing to the coast the victim decided to go in search of scallops. He was rowed out in a dinghy by a relative, whose other duty was to follow his progress by the bubbles. The first place was unsatisfactory so he towed a little further before diving again. There was a chop so the bubbles were impossible to identify. After waiting about one hour the man in the boat became anxious and returned to the shore, though it was a further one and a half hours before sufficient alarm was felt to notify the police. When the body was recovered, two days later, all equipment was intact and one hand was clutching the scallop bag, the other his regulator. He had not inflated his compensator, apparently.

NEW ZEALAND DIVING FATALITIES 1983

CASE	AGE	SKILL VICTIM	SKILL BUDDY	DIVE GROUP	DIVE BASE	WATER DEPTH DIVE	WATER DEPTH INCIDENT	WT ?	BELT WT	CONT. GAUGE	BUOY VEST	EQUIP TEST	REMAIN AIR	EQUIP. OWNER	WET SUIT	COMMENTS
BH 83/1	21	T Nil E Inex	N/S N/S	2	beach	> 6ft	surface	on	N/S	N/A	no	N/A	N/A	borrowed	yes	Strong swimmer. 3rd use snorkel. Diving for reef mussels. Incoming tide. No fins. No belt release. Tired buddy.
BH 83/2	16	T no	yes	2	beach	45ft	surface	on	N/S	N/A	no	N/A	N/A	borrowed	yes	Epileptic. Short separation. 2nd dive. Too deep for buddy to dive.
BH 83/3 (SC ??)	29	T N/S E yes	yes yes	2 sepn	rocks	70ft ??	N/S	on	N/S	N/A	off	N/A	N/A	borrowed	yes	Separation. Took off vest and tank. No quick release for weight belt. Hyperventilation?
BH 83/4	25	T N/S E Expd	N/S N/S	2 sepn	beach	12ft	N/S	on	N/S	N/A	no	N/A	N/A	own	yes	Separation. Hyperventilation? Adverse medical history
SC 83/1	21	T part E some	N/A	alone	river bank	15ft	N/S	on	N/S	N/A	no	N/A	N/A	own	yes	Alone. Alcohol. Incompletely trained.
SC 83/2	32	T yes expd	yes expd	2	boat	35ft	35ft	on	331b	yes	yes	yes	near full	own	yes	Crayfishing. Water power.
SC 83/3	34	T no E Inex	N/A	alone	boat	N/S	N/S	on	N/S	N/S	yes	yes	N/S	borrowed	N/S	Diving for scallops. Alone. Inexperienced Untrained.
SC 83/4	32	T N/S E some	N/S expd	3 sepn	beach	15ft	15ft	N/S	N/S	N/S	no	yes	nil	N/S	yes	Out of practice. 3rd in line. Separation. Poor visibility. Aspiration of vomit.
SC 83/5	46	T yes E inex	N/S N/S	3 sepn	boat	85ft	N/S	on	N/S	yes	infl. self	yes	nil	own	yes	Diving for scallops. Separation. Unconscious after surfaced. Weight belt twisted undroppable. Inexperienced. Air embolism.
SC 83/6	30	T pupil E inex	pupil inex	2	boat	N/S	surface	on	201b	yes	infl. buddy	yes	full	div. instr.	yes	Surface snorkel, full equipment and heavy belt. Inexperienced. Valiant buddy.

KEY T = Scuba course instruction
E = Experience of dive mode
N/S = Not Stated
N/A = Not Applicable

UNTRAINED, LACKING IN EXPERIENCE, ALONE, USING BORROWED EQUIPMENT.

Case SC 83/4

Intended as a friendly gesture, a refresher dive after a three year break from diving, it ended tragically. The diving history of the victim is unknown, as is the source of the equipment. Because it was only to be a "reminder" rather than a "serious" dive, it was undertaken in water no deeper than 25 ft, and at the critical time the three divers were proceeding in line ahead in 15 ft deep water, the victim bringing up the rear. His absence was noticed and an immediate search instituted, but poor visibility resulted in a short delay before he was found. Resuscitation efforts were unavailing. The tank was empty when tested. He did not have a "compensator" and had not dropped his weight belt. The autopsy gave drowning as cause of death, the aspiration of vomit noted being ascribed to the attempts made to resuscitate him.
UNKNOWN TRAINING, UNKNOWN EXPERIENCE. OUT OF PRACTICE. POOR VISIBILITY. INCORRECT BUDDY PROCEDURE. OUT OF AIR. SHALLOW WATER.

Case SC 83/5

This was a launch trip to a scallop bed organised for members of two diving clubs. The skipper assumed that responsibility for monitoring of the diving rested in their hands. A non-diver on board was co-opted as keeper of the log of diver water entry/exit times. Some of those present on the trip decided the locality was too deep for them (80-90ft) and did not dive, but the victim (newly certificated) and two others entered the water. It is apparent that there was no attempt to follow buddy-diving procedures. He was seen to surface, wave for attention, then float unconscious. His CO_2 type vest was seen to be inflated and the tank contents gauge showed "empty". He was rapidly brought on board but failed to respond to resuscitative measures. Autopsy showed that he had suffered an air embolism death. In retrospect those present agreed that he would not have been allowed to dive had his inexperience been realised.
NEWLY TRAINED. GROSSLY INEXPERIENCED. OUT OF AIR DESPITE HAVING GAUGE. ABSENCE OF DIVE DISCIPLINE & DIVING ORGANISATION OF OUTING. WEIGHT BELT TWISTED ROUND.

Case SC 83/6

The lectures and pool work of the course had been completed and the seven trainees were to make their first open water swim courtesy of the dive club whose boat trip they were permitted to join. Their test was a surface swim wearing full scuba equipment, including 20 lb weight belt, using a snorkel. After swimming half way to the marker rock, the victim held up the oral inflation tube of his compensator as if he was in trouble. His buddy inflated the vest orally and called for help. The instructor saw what was happening and quickly swam over to give assistance, starting EAR while towing the victim, by now unconscious. He was apparently still alive when unloaded from the boat into a waiting ambulance but died shortly afterwards. It was surmised that death resulted from aspiration of a little water, this causing an acute cardiac arrhythmia.

The resuscitation had caused fractured ribs, a reminder of the NECESSITY for correct resuscitation methods.
SURFACE SWIM. UNDER INSTRUCTION. FELLOW PUPIL, EXCELLENT BUDDY INSTRUCTOR AID. PRESUMED WATER ASPIRATION REACTION. RIB FRACTURES FROM RESUSCITATION.

DISCUSSION

Inexperience, undroppable weight belts, separation from buddies and the borrowing of equipment are adverse factors previously generally recognised as potentially lethal. Hyperventilation by breath-hold divers is another well documented danger. Cases BH 83/1 and SC 83/4 illustrate the poor basis for any belief that "it's only shallow" is a guarantee of safety. It is a commonplace to reiterate the conclusion that most fatalities are the end result of a number of safety violations, and therefore avoidable. Training, experience, respect for the water conditions, buddy diving discipline, having and taking notice of a contents gauge, and an effective buoyancy aid remain basic requirements for safe diving. Those who organise dive boat outings should accept the responsibility to supervise the safety of those present, or one day a court may remind them of their liability. The sea has more power than any diver, however physically fit and experienced he may be, and must always be included in consideration before entering the water.

ACKNOWLEDGEMENTS

This report could not have been compiled without the active assistance of the Water Safety Council of New Zealand and the New Zealand Department of Justice. Their interest in this Diving Safety Project is greatly appreciated.

INCIDENT REPORTING

Divers greatly benefit from the ongoing assessment, favourable and otherwise, of the experiences of others. Such information, collected and used on a basis of STRICT CONFIDENTIALITY regarding the identity of those involved, allows the early recognition of both helpful and dangerous diving practices. Reports, in particular concerning the successful management of diving-related misadventures and problems, are urgently required. New Zealand readers please support the Incident Reporting Scheme of the NZUA or write direct to:

Dr Douglas Walker
PO Box 120
NARRABEEN NSW 2101
Australia

YOU CAN'T PATENT THE WHEEL .... IF IT'S IN A CARTOON!

The NEW SCIENTIST reports that a large German chemical company tried to patent a way to raise sunken ships by pumping plastic balls into them. This would have been simpler than having to make them airtight and filling them with air. However the West German patents office screens all applications for new patents and turned down this one on the grounds that the idea had been used in a Wait Disney Donald Duck cartoon, tennis balls being used in this instance.

Reprinted from the Australian, 10 December 1983.

ARTERIAL GAS EMBOLISM AS A CONSEQUENCE OF PULMONARY BAROTRAUMA

Arterial gas emboli may result from arterialisation of venous gas emboli or may be introduced directly into the arterial circulation.[1] Included amongst the latter are divers, submarine escapees, and aviators who develop arterial gas emboli as a consequence of decompression in the absence of a tissue inert gas load that could account for gas emboli production.

THE ORIGINS OF ARTERIAL GAS EMBOLI COMPLICATING DECOMPRESSION

The conventional explanation is that decompression may result in an expansion of intrapulmonary gas in accordance with either Boyle's Law or Van der Waal's equation, sufficient to cause such pulmonary derangement that gas is introduced into the pulmonary vein.[2]

Despite 100 years of research the typical pulmonary lesion underlying the evolution of an arterial gas embolus has not been described. This makes it very difficult to prepare a suitable animal model to study subsequent events.

Few casualties with overt pulmonary damage due to decompression (pulmonary barotrauma) develop evidence of arterial gas embolism, and conversely few casualties suffering from arterial gas embolism have overt evidence of pulmonary damage. In the last 10 years at the Royal Australian Navy School of Underwater Medicine, 21 casualties have been treated for arterial gas embolism and 42 casualties have been treated for overt pulmonary barotrauma. During that time only one casualty has had unequivocal evidence of both arterial gas embolism and pulmonary barotrauma. This negligible overlap is surprising if these two conditions have a causal relationship.

TABLE 1
ROYAL AUSTRALIAN NAVY SCHOOL OF UNDERWATER MEDICINE
SURVEY OF GAS EMBOLISM AND PULMONARY BAROTRAUMA 1975-1983

Arterial Gas Embolism Cases	21
Pulmonary Barotrauma Cases	42
Combination of Gas Embolism and Pulmonary Barotrauma Cases	1

THE BEHAVIOUR OF GAS INTRODUCED INTO THE ARTERIAL CIRCULATION

The distribution of gas introduced into the arterial circulation is dictated by the posture of the diver, submarine escapee, aviator or experimental animals.[3] This explains both the preponderance of neurological involvement in divers and submarine escapees due to their upright posture, and the insistence on maintaining a casualty with arterial gas embolism in the head down/feet up posture.

The cerebrovascular reaction to a gas embolus differs from that to a solid embolus where vasoconstriction is usual, in that the reaction to a gas embolus is generally localised vasodilation.[5,6,7] A significant proportion of gas entering the cerebral circulation will pass through to the venous circulation without causing vessel occlusion.[3,5] Only when gas coalescence occurs to form a gas plug of sufficient size does vascular occlusion occur.[8] This is usually at a vessel bifurcation and in a vessel of 30-60 microns diameter.[8,9] The most plausible explanation of this is that the gas embolus will become stationary when the frictional forces that exist between the gas and the vessel exceed the local systemic driving pressure.[8]

THE PATHOLOGICAL SEQUELAE OF GAS EMBOLUS ENTRAPMENT

Once arterial gas emboli cause vascular occlusion, pathological processes are initiated. For the cerebral circulation these have been well defined.[2,5,8,9,17]

TABLE 2
PATHOLOGICAL SEQUELAE OF CEREBRAL ARTERIAL GAS EMBOLUS ENTRAPMENT

Increased blood-brain-barrier permeability

Tissue ischaemia

Downstream vessel coagulopathy

Small focal haemorrhages

Loss of cerebrovascular autoregulation

Metabolic disruptions

An increase in blood brain barrier permeability will result in an increase in brain water content, that is to say brain oedema of a vasogenic nature.[10,12] The rate of development of this brain oedema is critical to a major controversy in the treatment of cerebral arterial gas embolism. The rate of oedema accumulation following gas embolus entrapment is dependent upon whether the experimental animal is recompressed.[18] Human data is not available. If the animal is untreated, then oedema accumulates over several hours, but most studies have displayed negligible oedema within the first hour after embolisation.[10-12] Treatment of the animal by recompression appears to either prevent or retard the development of oedema.[18]

Other sequelae include tissue ischaemia which may result in infarction,[2,5,11,13] endothelial oedema and formation of platelet thrombi causing progressive vessel occlusion,[8,14,15] small focal haemorrhages,[9] and cellular metabolic disruptions.[11] The reported loss of vascular auto regulation has not been established experimentally. The vasoreactivity of a vessel adjacent to a gas embolus is unknown and is an area of active research within the Royal Australian Navy because of the current recommendation to use vasoconstricting gas mixtures.[19] If the vessel adjacent to a gas embolus is vasocreactive then vasoconstricting gases should initially be avoided as any vasoconstriction will

inhibit redistribution of the embolus by increasing both the gas-vessel interface and the frictional forces that oppose embolus movement.

PHENOMENA THAT MAY CONTRIBUTE TO THE CEREBRAL LESION

Any underlying pulmonary lesion and any cardiac involvement may contribute to cerebral pathology. While most theorists propose that pulmonary barotrauma underlies the evolution of arterial gas emboli, they also assume that the pulmonary lesion does not contribute to the evolving cerebral pathology. Possible ways in which any pulmonary lesion might contribute to cerebral pathology include systemic hypoxia, release of vasoactive substances stores in the lungs, impairment of venous return by expanding mediastinal gas, and most importantly, by continuing to introduce gas into the pulmonary vein. If any communication exists between the airways and the pulmonary vein, continued embolisation will result as a consequence of the pressure differential between them.[3]

THE EFFECTS OF RECOMPRESSION

Given a surface tension of 47 dynes/cm for plasma, thermodynamic theory predicts, for a typical gas embolus occupying a vessel of 30 to 60 microns diameter, that compression from 1 Ata to 6 Ata will not force the gas embolus into solution.[20] Nevertheless if a casualty with a cerebral arterial gas embolus is recompressed with minimal delay from onset of symptoms and signs, as in the Submarine Escape Training Tank (SETT) at HMS DOLPHIN, almost all casualties achieve dramatic total relief.

TABLE 3
TIMES FROM THE ONSET OF COMPRESSION TO TOTAL RELIEF OF SIGNS AND SYMPTOMS

Time in Minutes	Cases
less than 1	42
1 - 5	30
6 - 30	8
31 - 120	2

In the 112 cases of cerebral arterial gas embolism recorded at HMS DOLPHIN the time from the start of compression to the time of total relief was determined where possible. For 20 cases this was not recorded clearly. Three cases obtained no relief and died. Another 7 cases obtained total relief only after decompression from 50 msw. The remaining 82 cases obtained total relief before decompression.

Such rapid total relief can only be explained by redistribution of the gas embolus as a result of reduced gas-vessel interface frictional forces.[6,8,9] Of note is the finding that after recompression of embolised rats to 6 Ata, 30-40% of the gas redistributed to other parts of the brain.[6] This is one mechanism that can explain those casualties who relapse with different symptoms and signs from their initial presentation.

THE PATHOLOGY OF RELAPSE

Following the dramatic recovery of nearly all casualties treated without delay, some casualties either relapse or deteriorate. For most groups studied, whether they be submarine escapees or divers, the proportion relapsing is about 30%. Of this group about 5 to 10% die, contributing to the overall mortality of cerebral arterial gas embolism which even in the best circumstances is about 5%.[14,19,21,31]

Numerous pathologies have been proposed to explain these casualties that relapse or deteriorate.[2,4,6,8,10-12,14,15,21,30,31] Re-embolisation may result from redistribution of a cerebral arterial gas embolus to another region of the brain, redistribution of gas from elsewhere in the body to the cerebral circulation, for example, with a change of posture, or as a consequence of continued embolisation from an original pulmonary lesion.[32]

TABLE 4
PROPOSED CAUSES OF RELAPSE OR DETERIORATION AFTER RECOMPRESSION

Re-embolisation

Regeneration of gas volume

Focal brain oedema

Failure of re-perfusion

Others

Careful examination of the literature and casualty case reports indicates that no single proposed cause of relapse can explain the majority of cases that relapse or deteriorate after initial improvement[2,4,6,8,10-12,14,15,19,21-31]. This contrasts with the recent proposal that brain oedema is responsible for the majority of cases that relapse and that treatment of a relapse should consist of increased “brain-oedema” therapy and not recompression.[19,21,30,31] Much of the evidence for this proposal comes from case report symptomatology.[30,31] However, brain oedema is a pathological and not a clinical diagnosis, as there is no symptom complex indicative or typical of brain oedema.[32]

There is other evidence against brain oedema as the major cause of relapses. First, brain oedema is related to the original site of the cerebral arterial gas embolus and cannot account for those casualties that relapse with totally different symptoms from the original presentation (Table 10).[19] Second, the rate at which brain oedema develops and resolves is incompatible with those casualties that relapse dramatically during decompression and respond immediately to recompression. This phenomenon is demonstrated by the data from the SETT at HMS DOLPHIN displayed in Table 5. These were 32 casualties of whom two relapsed twice so giving a total of 34 relapses.

Note that 14 cases relapsed either during or within 5 minutes of decompression to a shallower depth. Of the 16

TABLE 5
ONSET OF RELAPSES AND THE RESPONSES OF RELAPSES RECOMPRESSED IMMEDIATELY

Time of onset of relapse or deterioration	While at 50m	During decompression to a shallower depth	Minutes after arrival at a shallower depth					
			0-1	1-5	6-11	11-15	16-30	More than 30
Number of relapse cases	5	6	3	5	4	1	3	7
Number of cases recompressed immediately after a relapse	0	3	3	5	3	0	0	2
Number obtaining immediate total relief with recompression	-	2	1	1	0	-	-	1
Number obtaining total relief within 10 minutes of recompression	-	0	0	3	3	-	-	0
Number obtaining total relief more than 10 minutes after recompression	-	1	1	1	3	-	-	1

Five cases relapsed or deteriorated while the casualty was at 50msw. The remaining 27 cases relapsed or deteriorated during or after decompression. Two cases relapsed twice giving the total of 34 relapses in this table. Sixteen of the 27 cases were recompressed as soon as the symptoms or signs of the relapse or deterioration became apparent. The responses to recompression are also detailed.

TABLE 6
TIME FROM ONSET TO RELAPSE IN HOURS

Hours from Onset to Relapse	No. Cases
Less than 1	14
1 - 2	5
2 - 3	2
3 - 4	1
4 - 5	1
5 - 6	1
6 - 7	0
Greater than 7	2

In 6 of the 32 cases of relapse the time from onset to relapse was not clearly recorded.

TABLE 7
TIME FROM ONSET TO RELAPSE IN MINUTES

Hours from Onset to Relapse	No. Cases
Less than 20	2
20 - 40	9
40 - 60	3
60 - 90	3
90 - 120	2

These cases appear in the "less than 1 hour" and "1 to 2 hours" classifications in Table 7.

casualties who relapsed and were recompressed within 5 minutes of the onset of the relapse, 8 obtained total relief from the relapse within 10 minutes of recompression and a further 7 obtained relief which was complete more than 10 minutes after recompression. Another 6 cases not recorded in Table 5 were recompressed after they relapsed, but recompression began more than 5 minutes after the onset of the relapse. Of this group, 5 recovered rapidly after recompression and 1 recovered gradually but eventually completely.

Third, as discussed, even for untreated experimental animals negligible brain oedema accumulates during the first hour after embolisation.[10-12,18] It follows that brain oedema cannot account for the peak occurrence of relapses and deteriorations at 20 to 40 minutes after the initial presentation for the 32 casualties that have relapsed or deteriorated after initial improvement at HMAS DOLPHIN.

Clearly brain oedema is a complication of untreated cerebral arterial gas embolism. However it does not provide a good explanation of the majority of cases that relapse or deteriorate, and consequently "brain-oedema" therapy is not an adequate response to all relapses or deteriorations. There are numerous possible causes of relapse or deterioration after initial improvement and the clinical response to a relapse or deterioration must be dictated by both the circumstances in which the relapse occurred and by the nature of the relapse.

THE CLINICAL PRESENTATION OF CEREBRAL ARTERIAL GAS EMBOLISM

The symptomatology from the HMS DOLPHIN cerebral arterial gas embolism cases are typical of other reports and are listed at Tables 8 and 9.[4,28,29]

TABLE 8
PRESENTING SYMPTOMS AND SIGNS OF 112 CASES OF CEREBRAL ARTERIAL GAS EMBOLISM

Unconsciousness	45
Sensory loss	25
Paresis	24
Stupor	22
Collapse without unconsciousness	13
Visual disturbances	10
Convulsions	4

TABLE 9
PRESENTING SYMPTOMS AND SIGNS OF THE 32 CASES THAT RELAPSED AFTER INITIAL IMPROVEMENT

Unconsciousness	14
Sensory loss	5
Paresis	7
Stupor	7
Collapse without unconsciousness	4
Visual disturbances	2
Convulsions	1

Note the common symptom frequency distribution for all cases and for those cases that relapsed and that it is therefore impossible to predict which cases will relapse or deteriorate on the basis of their presenting symptoms and signs.

In contrast, for the casualties that relapsed, the symptom frequency distribution at initial presentation and at relapse are different.

TABLE 10
SYMPTOMS AND SIGNS ON PRESENTATION AND RELAPSE

Symptoms and Signs	Initial Presentation	Relapse
Unconsciousness	14	2
Sensory loss	5	7
Paresis	7	7
Stupor	7	6
Collapse without unconsciousness	4	0
Visual disturbances	2	9
Convulsions	1	6

32 patients relapsed following improvement. Some casualties presented with more than one symptom or sign, either initially or on relapse.

This is relevant to focal pathologies proposed as causes of relapse or deterioration. Any pathology that is related to the original site of the cerebral arterial gas embolus such as brain oedema, regeneration of gas volume, and failure of reperfusion due to progressive vascular occlusion should result in a relapse that is similar to the initial presentation.

GENERAL COMMENTS ON THE TREATMENT OF CEREBRAL ARTERIAL GAS EMBOLISM

Most discussions of the treatment of cerebral arterial gas embolism do not differentiate between SETT casualties and divers. This a major oversight as the two groups are significantly different.[33] SETT casualties have a negligible increase in tissue inert gas content. In contrast divers have a variable but significant increase, particularly when it is remembered that the half life for uptake of inert gas by the brain is only 5 minutes. The inert gas elimination kinetics are totally different for these two situations. Cerebral arterial gas embolism is most frequently encountered in sports divers, who, rarely having a recompression chamber at their diving site, rarely present for treatment within 1 hour, in contrast to SETT casualties who are recompressed immediately their symptoms develop.

These observations illustrate the difference between SETT casualties and divers with cerebral arterial gas embolism, the inappropriateness of combining data from divers and SETT casualties as has been done by many authors,[30,31,34] and why the Royal Australian Navy is developing different animal models to evaluate the treatment needs of these two situations.

Another major treatment concept is that any delay in recompression will result in increased morbidity and mortality.[19] There are no controlled human studies to support this. Sufficient numbers of otherwise homogeneous cases for comparison can only be generated by review of SETT casualty case reports. In this assessment the relapse rate is used as the indicator of success or failure. Although the data displayed suggests that any delay may lead to a worse outcome, the difference displayed for these two groups is too small to establish statistical significance. Nevertheless it is reasonable to accept that until better data is available the time from onset of symptoms to recompression should be minimised.

TABLE 11
THE RELATIONSHIP OF DELAY IN COMPRESSION OF THE CASUALTY TO 50 MSW TO RELAPSE

Time from onset of symptoms to arrival at 50 msw in minutes	Total Number	Relapse Number	Relapse Rate
Less than 582	22	27%	
5 or more 19	7	37%	

The time interval from the onset of symptoms and signs to compression of the casualty to 50 metres was evaluated in all 112 cases. For 1 case the time taken to reach 50 msw was not recorded clearly. This casualty did not relapse. Another 7 cases were compressed to depths other than 50 msw. Three of these 7 cases relapsed. Three other cases showed no improvement with recompression and died. The remaining 101 cases are detailed above.

THE TREATMENT DEPTH

Current regimens advise compression to 50 msw (165 ft) if treatment is instituted within five hours.[35,36] This is

based on the following. First, although a cerebral arterial gas embolism becomes more spherical with volume reduction, the rate of reduction in gas diameter decreases markedly with increasing depth such that beyond 50 msw considerable further compression is required to produce any noticeable reduction in gas embolus diameter. This does not apply to the same degree to any gas embolus which remains in a cylindrical configuration. Second, animal studies have demonstrated that cerebral arterial gas emboli redistribute with compression to 50 msw, with some embolus redistribution seen at 30 msw (100 ft).[9] Third, significant human data only exists for treatment at 50 msw.[1,37] For example only 7 of the 115 cases from HMS DOLPHIN were not treated at 50 msw. All 7 were treated at shallower depths than 50 msw and 3 of these cases relapsed.

Leitch et als study of embolised rats at NMRL suggests that treatment at 18 msw (60 ft) breathing oxygen might be as good as compression to 50 msw.[18] The study findings conflict with an unpublished study by Bayliss, who decompressed rats with ligated tracheas and have the serious limitations of any study that uses the technique of direct infusion of gas into the vessels supplying the cerebral circulation. However it does provide support for the widely accepted proposition that if a casualty presents after 5 hours he should only be compressed to 18 msw[35,36] and also provides additional stimulus for the Royal Australian Navy's current evaluation of different treatment depths.

TIME AT THE TREATMENT DEPTH PRIOR TO DECOMPRESSION

During the last 15 years, despite the reports of Elliott, Harrison et al[28] and Ah-See,[4] an increasingly common practice has been to begin decompression from 50 msw after 30 minutes if the casualty is asymptomatic.[9,34,38-40] However comparison of the relapse rates of otherwise homogeneous SETT casualties and the time spent at 50 msw prior to decompression indicates that this practice should be abandoned.

The 16 to 30 minute group was comprised of casualties who were kept at 50 msw for 30 minutes prior to decompression, The 31 to 120 minute group included 8 casualties who were kept at 50 msw for 60 minutes, while the remainder were kept at 50 msw for 2 hours prior to decompression. By X^2 analysis, the relapse rates for these groups are significantly different at the 0.01 level.

Clearly casualties kept at 50 msw for only 30 minutes have a significantly greater relapse rate than those kept at 50 msw for either 1 or 2 hours. Decompression from 50 msw should not begin after 30 minutes, and probably a casualty should be kept at 50 msw for as long as possible while still maintaining access to a conventional therapeutic table. If the therapist has access to therapeutic tables such as Royal Navy Table 54, then 2 hours at 50 msw would appear indicated.

OXYGEN AT HIGH PRESSURES

Since the introduction of the Goodman and Workmann oxygen breathing therapeutic tables,[41] the concept that "the more oxygen you give, the better you are doing" has

TABLE 12
THE RELATIONSHIP BETWEEN TIME SPENT AT 50 MSW BEFORE DECOMPRESSION AND RELAPSE

Time at 50 msw Prior to Decompression (Min)		Total Number	Relapse Number	Relapse Rate
0 - 15	18	8	44%	
16 - 30	34	12	35%	
31 - 120	28	2	7%	

The time spent at 50 msw prior to decompression was considered for all 112 cases. This time was not clearly recorded in 17 cases, 7 of which subsequently relapsed. Another 7 casualties were recompressed to depths other than 50 msw; 3 of these 7 cases relapsed. Three cases showed no improvement with recompression ad died. Five casualties relapsed at 50 msw prior to decompression. The remaining 80 cases were analysed and are presented above.

crept into underwater medicine folk-law. This has resulted in attempts to increase the inspired partial pressure of oxygen being breathed at 50 msw in the treatment of cerebral arterial gas embolism.[19] It is important to review the reported advantages and disadvantages of breathing oxygen at high pressure.

TABLE 13
REPORTED ADVANTAGES OF OXYGEN AT HIGH PRESSURES

Reduced cerebral blood flow
Increased tissue oxygenation
Reduced blood brain barrier permeability
Positive vascular steal

Reported advantages of oxygen at high pressures include cerebral vasoconstriction resulting in a fall in cerebral blood flow, a fall in intracranial pressure, and therefore increased rate of brain oedema resolution, increased tissue oxygenation, and decreased blood brain barrier permeability.[17,42-44]

TABLE 14
REPORTED DISADVANTAGES OF OXYGEN AT HIGH PRESSURES

Inert gas
Oxygen toxicity
Rebound in brain oedema following withdrawal
Increased blood brain barrier permeability Increased cerebral blood flow

Reported disadvantages of oxygen at high pressure include the observation that if oxygen is supplied in excess of local metabolic needs it will behave as an inert gas, pulmonary and CNS oxygen toxicity, increased blood brain barrier permeability, increased cerebral blood flow resulting in increased intra-cranial pressure, impaired cerebral glucose metabolism, and gas embolus expansion.[5,43,45-52]

The apparent contradictions result from the observation that alteration of the inspired partial pressure of oxygen

can result in a considerably different effect.[42,44,52] If the inspired partial pressure of oxygen is in the 1 to 1.5 ATA range cerebral vasoconstriction, improved cerebral glucose metabolism, and improved EEG recordings are the usual findings. If however the inspired partial pressure of oxygen is increased to and beyond 2 Ata, both cerebral glucose metabolism and EEG recordings deteriorate.[50-52] Also in this range the effects on cerebral blood flow are variable. A particularly common finding is for an immediate increase in cerebral blood flow with a fall towards the previous level over 30 to 40 minutes.[50,51]

TABLE 15
THE EFFECTS OF RAISING THE INSPIRED PARTIAL PRESSURE OF OXYGEN

P_i0_2 in ATA	Glucose Utilization	EEG	Cerebral Blood Flow
1	↑	Improved	↓
1.5	↑	Normalised	↓
2	↓	Deteriorated	↑↓
2.8	↓	Grossly Abnormal	↑↓

It could be argued from this data that the inspired partial pressure of oxygen be maintained within the 1 to 1.5 Ata range. Conveniently this is achieved by breathing air at 50 msw avoiding the need to use a built-in breathing system. The only indication to exceed this range would be to increase the elimination of inert gas. This is not relevant to the SETT casualty but may be relevant to the diver, and is yet another phenomenon being investigated by the Royal Australian Navy. A reasonable regimen would be to administer an inspired partial pressure of oxygen of 1 to 1.5 Ata to SETT casualties, and until further information is available to adjust the inspired partial pressure given to a diver according to his dive profile.

CHEMOTHERAPY FOR CEREBRAL ARTERIAL GAS EMBOLISM

There are no drugs of proven benefit for the primary problem of a gas embolus occluding a vessel although research into the role of heparin, indomethacin and prostaglandin 12 in facilitating reperfusion of the brain after embolus redistribution is having encouraging results. These drugs may become important adjuvants to recompression.[53-58] The use of naloxone and dimethyl-sulphoxide (DMSO) also have support from experimental animal studies.[59,60]

Most forms of chemotherapy suggested for use in the treatment of cerebral arterial gas embolism relate to either preventing or ameliorating the development of brain oedema.[2,13,21,32,48,61-64] The inability to predict which cases will relapse on the basis of their presentation, and the lack of an identifiable brain oedema clinical syndrome, result in the inevitable conclusion that if a prophylactic brain oedema regimen is indicated, it should be given to all casualties.

Although the initial brain oedema complicating cerebral arterial gas embolism is vasogenic, eventually both vasogenic and cytotoxic brain oedema will be present.[13] This places another condition on a brain oedema regimen in that it must include agents active against both forms of oedema. Non-osmotic diuretics, such as furosemide, dexamethasone and phenobarbitone have been shown to be effective in treating vasogenic brain oedema models.[13,32,62,64] Osmotic diuretics, such as mannitol and glycerol, non-osmotic diuretics and methylprednisolone sodium succinate have been shown to be effective in treating cytogenic brain oedema models.[2,13,32,64]

A recent report of the efficacy of a steroid regimen in reducing the relapse rate associated with cerebral arterial gas embolism inappropriately combined diving casualties and SETT casualties to generate sufficient numbers for comparison.30,31 The efficacy of a steroid regimen in treating brain oedema consequent on a cerebral arterial gas embolus is yet to be established. It must be noted that steroid regimens for other causes of brain oedema are falling into disrepute.32,64 It should also be noted that given the wide range of steroidal effects the efficacy or lack of efficacy of asteroid regimen does not implicate or refute any proposed underlying pathology.30,31

Other areas of treatment of cerebral arterial gas embolism include the management of a relapse or deterioration mentioned previously, the possible roles of mechanical ventilation to maintain hypocarbia and in particular high frequency ventilation (HFV), and hypothermia in the management of the critically ill casualty.32,48

CONCLUSIONS

Far from being an area of consensus and understanding, cerebral arterial gas embolism is an area of confusion and active debate. The confusion relates not only to the underlying pathological processes but also to the ideal treatment of cerebral arterial gas embolism affecting divers, submarine escapees and aviators.

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REFERENCES

1. Hart GB. Treatment of decompression illness and air embolism with hyperbaric oxygen. Aero*sp Med*. 1974; 45: 1190-1193

2. Peirce EG. Cerebral gas embolism (arterial) with special reference to iatrogenic accidents. *Hyperbaric Oxygen Review*. 1980; 1: 161

3. Van Allen CM, Hrdina LS, Clark J. Air embolism from the pulmonary vein. *Arch Surg*. 1929; 19: 567-599

4. Ah-See AK. Review of arterial air embolism in submarine escape. In: Smith G (ed). *Proceedings of the 6th International Congress on Hyperbaric Medicine*. Aberdeen: Aberdeen University Press, 1979; 349-351

5. de la Torre E, Meredith J, Netsky M. Cerebral air embolism in the dog. *Arch Neurol.* 1962; 6: 307-316

6. Grulke DC and Hills BA. Experimental cerebral air embolism and its resolution. In: Shilling CW and Beckett MW (eds). *Proceedings of the 6th Underwater Physiology Symposium*. Washington: FASEB, 1978; 587-594

7. Hills BA. Scientific consideration in recompression therapy. In: James PB, McCallum RI and Rawlins JSP (eds). *Proceedings of the 7th Annual Congress of the European Undersea Biomedical Society*. Norwich: Norwich Union, 1981; 143-162

8. Grulke DC. Experimental cerebral air embolism: a physical and physiological study using uniform microbubbles of known size. PhD Thesis, London: University of London, 1975

9. Waite CL, Nazzone WF, Greenwood ME, Larsen RT. Cerebral air embolism. I. Basic studies. US Navy Submarine Medical Centre Report No 493, 1967

10. Ah-See AK. Permeability of the blood-brain barrier to FITC labelled dextran in massive cerebral air embolism. In: Hallenbeck JM and Greenbaum LJ (eds). *Air Embolism and Acute Stroke*. Bethesda, Maryland: Undersea Medical Society Inc, 1977; 43-48

11. Kogure K, Busto R, Alonso OF and Samson R. Effects of recompression treatment on cerebral energy metabolism in arterial air embolism of the rat brain. In: Hallenbeck JM and Greenbaum LJ (eds). *Air Embolism and Acute Stroke*. Bethesda, Maryland: Undersea Medical Society Inc, 1977; 105-122

12. Nishimoto K, Wolman M, Spatz M and Klatzo I. Pathophysiologic correlations in the blood-brain-barrier damage due to air embolism. *Adv Neurol. 1978; 20*: 237-244

13. James HE. Combination therapy in brain oedema. *Adv Neurol* 1980; 28: 491-502.

14. Hallenbeck JM. Prevention of postischaemic impairment of microvascular perfusion. *Neurology,* 1977; 27: 3-10

15. Hallenbeck JM and Furlow TW Jr. Impaired microvascular perfusion and secondary deterioration in dysbaric cerebral air embolism. In: Hallenbeck JM and Greenbaum LJ (eds). *Air Embolism and Acute Stroke*. Bethesda, Maryland: Undersea Medical Society Inc, 1977; 76-86

16. Paulson OB. Cerebral apoplexy (stroke): pathogenesis pathophysiology and therapy as illustrated by regional blood flow measurement in the brain. S*troke 1971*; 2: 327-360

17. Marmarou A, Takagi H and Stulman K. Biomechanics of brain oedema and effects on local cerebral blood flow. *Adv Neurol* 1980; 28: 345-358

18. Leitch DR, Greenbaum LR Jr and Hallenbeck JM. Cerebral Arterial air embolism. Parts I-IV. Hyperbaric Medicine Program Centre, Naval Medical Research Institute. Bethesda, Maryland. In preparation.

19. Pearson RR. Treatment of submarine-escape-training casualties. In: *Minutes of the Submarine Escape and Rescue Workshop.* HMS DOLPHIN, 1979; C60-C63

20. Weathersby PK, Homer LD, Flynn ET. Homogenous nucleation of gas bubbles in vivo. *J Appl Physiol: Respirat Environ Exercise Physiol. 198*2; 53(4): 940-946

21. Pearson RR. Submarine escape research. In: *Proceedings of the Submarine Medicine Conference.* Institute of Naval Medicine, 1980; 22-24

22. Behnke AR. Analysis of accidents occurring in training with the submarine "lung". *US Nav Med Bull.* 1932; 30: 111-185

23. Polak B and Adams BH. Traumatic air embolism in submarine escape training. *US Nav Med Bull.* 1932; 30: 165

24. Peirano JH, Alvis HJ and Duffher GJ. Submarine escape training experience 1929-1954. *USN Medical Research Laboratory Report 264*, 1955

25. Kinsey JL. Air embolism as a result of submarine escape training *US Armed Forces Med J.* 1956; 5: 243-255

26. Moses H. Casualties in individual submarine escape training. *USN Submarine Medical Centre Report No 438.* 1964

27. Ingvar DH, Adolfson D and Lindemark C. Cerebral air embolism during training of submarine personnel in free escape. *Aerosp Med* 1973; 44: 635-638

28. Elliott DH, Harrison JAB and Barnard EEP. Clinical and radiological features of 88 cases of decompression barotrauma. In: *Proc of the 6th Underwater Physiology Symposium.* In Shilling CW and Beckett MW (eds). Bethesda, Maryland: FASEB, 1978; 527-535

29. Greene KM. Causes of death in submarine escape training casualties: analysis of cases and review of the literature. *AMTE(E) Report R78-402*, 1978

30. Pearson RR. Aspects of pulmonary barotrauma The aetiology, pathophysiology, prevention and therapy of pulmonary barotrauma and arterial gas embolism resulting from submarine escape training

and diving. MD Thesis, University of Newcastle, 1982

31. Pearson RR, Goad RF. Delayed cerebral edema complicating cerebral arterial gas embolism: case histories. *Undersea Biomed Res*. 1982; 9(4): 283-296

32. Marshall LF. Treatment of brain swelling and brain oedema in man. *Adv Neurol*. 1980; 28: 459-470

33. Elliott DH. Introductory outline of dysbaric air embolism and the operational problems encountered. In: Hallenbeck JM and Greenbaum LJ (eds). *Air Embolism and Acute Stroke*. Bethesda, Maryland: Undersea Medical Society Inc, 1977; 1-5

34. Bornmann RC. Experience with minimal recompression oxygen breathing treatment of decompression sickness and air embolism. *USN EDU Project SF0110605 Task 11513-2 Memorandum Report, 1967*

35. European Undersea Biomedical Society. The choice of a therapeutic compression table in relation to the causative dive. London 1976

36. Barnard EEP. The practical implications of current research. In: Hallenbeck JM and Greenbaum LJ (eds). *Air Embolism and Acute Stroke*. Bethesda, Maryland: Undersea Medical Society Inc, 1977; 180-184

37. Workman RD. Treatment of bends with oxygen at high pressure. Aerosp Med 1968; 39: 1076-1083

38. Van Genderen L and Waite CL. Evaluation of the rapid recompression - high pressure oxygenation approach to the treatment of traumatic cerebral embolism. *US Navy Submarine Medical Centre Report No. 519,* 1968

39. Kindwall EP. Hyperbaric and ancillary treatment of decompression sickness air embolism and related disorders. In: *Diving Medicine*. Strauss RH (ed). New York: Grune & Stratton, 1976; 83-96

40. Goad RF. Potential embolism. In: *Decompression Sickness and its Therapy*. Lambertsen CJ (ed). Allentown, PA: Air Products Inc, 1978; 19-27

41. Goodman MW and Workman RD. Minimal recompression oxygen breathing approach to treatment of decompression sickness in divers and aviators. *US Navy Experimental Diving Unit Research Report 5-65*. 1965

42. Miller JD, Ledingham IMcA and Jennett WB. Effect of hyperbaric oxygen on intra-cranial pressure and cerebral blood flow in experimental cerebral oedema. *J Neurol Neurosurg Psychiat*. 1970; 33: 745-755

43. Lanse SB, Lee SC, Jacobs EA and Brody H. Changes in the permeability of the blood-brain-barrier under hyperbaric conditions. In: *Proceedings of the 6th International Hyperbaric Congress*. Smith G (ed). Aberdeen University Press: 1979; 101-103

44. Bassett BE and Bennett PB. Introduction to the physical and physiological basis of hyperbaric therapy. In: *Hyperbaric Oxygen Therapy*. Davis SC and Hunt TK (eds). Bethesda, Maryland: Undersea Medical Society Inc, 1977; 11-24

45. James PB. Problem areas in the therapy of neurological decompression sickness. In: *Proceedings of the 7th Annual Congress of the European Undersea Biomedical Society*. James PB, McCallum RI and Rawlins JSP (eds). Norwich: Norwich Union, 1981; 127-142

46. Bert P. *Barometric Pressure: Researches in Experimental Physiology*. G Masson, Paris: 1878. Transl. by Hitchcock M and Hitchcock FA. Columbus, Ohio: Longs College Book Co, 1943.

47. Smith JL. The pathological effects due to increase of oxygen tension in the air breathed. *J Physiol*. 1899; 24: 19-35

48. Peirce EC and Jacobsen CH. Cerebral oedema. In: *Hyperbaric Oxygen Therapy*. Davis SC and Hunt TK (eds). Bethesda, Maryland: Undersea Medical Society Inc, 1977; 287-301

49. Ledingham I McA. Hyperbaric oxygen. In: *Recent advances in Surgery*. Taylor S (ed). London: Churchill, 1969; 295-338

50. Holbach KH, Wassman H and Careli A. Continuous CBF measurements during hyperbaric oxygenation In: *Proceedings of the 6th International Hyperbaric Congress*. Smith G (ed). Aberdeen University Press: 1979; 104-111

51. Holbach KH, Wassman H and Careli A. Correlation between EEG and CBF changes during hyperbaric oxygenation In: *Proceedings of the 6th International Hyperbaric Congress*. Smith G (ed). Aberdeen University Press: 1979; 112-117

52. Holbach KH and Careli A. Oxygen tolerance and the oxygenation state of the injured human brain. In: *Proceedings of the 5th international Hyperbaric Congress*. Trapp WG, Banister WE, Davison AJ, Trapp PA (eds). Burnaby: Simon Fraser University, 1974; 350-361

53. Hallenbeck JM, Furlow TW, Ruel TA and Greenbaum LJ, Jr. Extracorporeal glass-wool filtration of whole blood enhances postischaemic recovery of the cortical sensory evoked response. *Stroke*. 1979; 10; 2: 158-164

54. Hallenbeck JM and Furlow TW, Jr. Prostaglandin 12 and Indomethacin prevent impairment of post-ischaemic brain reperfusion in the dog. *Stroke*. 1979; 10; 6: 629-637

55. Hallenbeck JM and Furlow TW Jr, and Gralnick HR. Influence of Factor VIII/von Willebrand Factor Protein (F VIII/VWF) and F VIII/VWF-poor cryoprecipitate on post-ischaemic microvascular reperfusion in the central nervous system. *Stroke*. 1981; 12; 1: 93-97

56. Hallenbeck JM, Leitch DR, Dutka AJ, Greenbaum LJ Jr, McKee AE. Prostaglandin 12 Indomethacin and Heparin Promote postischemic neuronal recovery in dogs. *Ann Neurol*. 1982; 12: 145-156

57. Hallenbeck JM. Hypervolemic Hemodilution in acute stroke *JAMA*. Dec 10 1982; 248: 22

58. Hallenbeck JM, Leitch DR, Dutka AJ and Greenbaum LJ Jr. The amount of circumscribed brain edema and the degree of post-ischaemic neuronal recovery do not correlate well. *Stroke*. 1982: 13; 6: 797-804

59. de la Torre JC, Surgeon JW, Hill PK and Khan T. DMSO in the treatment of brain infarction: basic considerations. In: Hallenbeck JM and Greenbaum LJ (eds). *Air Embolism and Acute Stroke*. Bethesda, Maryland: Undersea Medical Society Inc, 1977; 138-161

60. Faden AI, Hallenbeck JM, Brown CQ. Treatment of experimental stroke: Comparison of naloxone and thyrotropin releasing hormone. Neurology. 1982; 32: 1083-1087

61. Dunn JE. An evaluation of hyperbaric oxygen hypocapnic hyperventilation and methylprednisolone therapy in cold-induced cerebral swellings. In: *Proceedings of the 5th international Hyperbaric Congress*. Trapp WG, Banister WE, Davison AJ, Trapp PA (eds). Burnaby: Simon Fraser University, 1974; 370-382

62. Safar P, Stezoski W, Nemoto EM. Amelioration of brain damage following 12 minutes of cardiac arrest in dogs. Arch Neurol. 1976; 33: 91

63. Pearson RR. A review of the therapy of decompression illness. In: *Proceedings of the 6th International Hyperbaric Congress*. Smith G (ed). Aberdeen University Press: 1979; 392-399

64. Meinig G, Ruelen HJ, Simon RS and Schurmann K. Clinical chemical and CT evaluation of short-term and longterm antiedema therapy with dexamethasone and diuretics. *Adv Neurol*. 1980; 28: 471-490

Dr Gorman is a medical officer at the Royal Australian Navy School of Underwater Medicine. However the views expressed in this paper are not necessarily those of the Royal Australian Navy.

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HYPERBARIC OXYGEN FOR MULTIPLE SCLEROSIS

Philip James

The medical profession's response to the introduction of yet another therapy in multiple sclerosis is a scepticism conditioned by years of frustration in the search for a causative agent and an effective remedy.

To suggest that oxygen may be of help in multiple sclerosis (MS) would seem extremely farfetched, especially when the last 25 years have seen research effort into the immunological abnormality in MS, even though other diseases where the cause is known, for example neurosyphilis, produce similar changes. Over 47 studies of immunosuppression therapy, including several controlled trials, have failed to show clear evidence of benefit to patients.

January of this year saw a milestone in the history of MS with the publication of a successful double-blind, controlled trial.[1] The treatment group received oxygen under hyperbaric conditions. There was immediate improvement in 12 out of 17 of the treated group, contrasted with 1 out of 20 in the controls ($p<0.0001$).

Perhaps even more remarkable, there was stabilization of the 12 patients who had responded to the oxygen therapy over the subsequent year. Five maintained their improvement and none of the 12 had deteriorated to below the pre-treatment level. Of the five remaining patients in the treated group, who did not show objectively measurable improvement, only two showed deterioration over the following year. In contrast, with the control group, 11 of the 20 patients had deteriorated over this period yielding a p value of < 0.0008.

A favourable response to oxygen is by definition an indication of hypoxia and should re-direct our attention to evidence of blood vessel involvement in the disease. Typically, there are lesions in the cerebellum of patients with MS. Current immunological ideas would have us believe that these lesions and the accompanying grossly dilated vein are the result of an isolated focus of autoimmune activity in the surrounding tissue. Because of the abundant evidence that oxygen influences the cerebral vasculature in general, and the cerebral veins in particular, it is vital that we re-examine fundamental aspects of this disease.

Multiple sclerosis is, of course, not a diagnosis but a pathological description of the appearance of the brain at post-mortem examination. The suggestion that the disease is simply demyelination of fibres in the white matter may lead to the feeling that the condition is curable, but the loss of cells, fibres and the gliosis in lesions contradicts this. Established multiple sclerosis is simply a reference to multiple scars in the central nervous system and, as such, must represent an incurable condition. The preservation of fibres stressed by Charcot is never more than "relative" and Simpson has recently emphasized the importance of grey matter lesions in MS, indicating that they are required for the diagnosis. An immunological attack on myelin cannot account for this fibre destruction, nor can it account for lesions in the spinal cord, which sometimes produce

central infarction with preservation of some of the surrounding white matter. This pathology must be accommodated by any hypothesis of causation and not discarded because it is inconvenient.

It is most important to recognize that MS is unique in requiring multiple lesions to develop before a "diagnosis" based on more than one lesion can be made. The question must surely be is there a disease that should be called "monosclerosis"? Reference to the pathological literature indicates that single, silent plaques are a comparatively common finding at necropsy and may even be found in the spinal cord.

In view of neuropathological emphasis on the necessity for grey matter lesions to make a pathological "diagnosis" of MS, and the likelihood of such lesions being associated with disability, has the emphasis on white matter plaques been a red-herring? Every study of plaques and disability has shown that they do not correlate, yet despite this, researchers continue to be obsessed with plaques and even attempt to dissociate the "real" disease from the lesions causing the symptoms.[3] Part of this false trail has been to label the disease "demyelinating" and cause generations of researchers to ignore the constant destruction of some fibres in lesions.

BLOOD-BRAIN BARRIER DISTURBANCES

Both radio-isotopes in the 1960s and contrast-enhanced CAT in the 1970s have shown that the blood-brain barrier is disturbed in acute attacks. The extreme sensitivity of nervous tissue to the acute oedema resulting from this dysfunction is well known, and the oligodendrocytes, whose cellular processes form the myelin sheaths, are the cells most vulnerable, not the neurone itself. The damage occurs in the CNS within hours and, whatever the cause of this disease, the initial symptoms must be treated early to prevent permanent damage and disability.

In view of this, the results of the New York hyperbaric oxygen trial, in which severely affected chronic stable or chronic progressive patients with a minimum diagnosed disease duration in excess of five years were chosen, are remarkable.

An agent found to be of benefit in the advanced disease must surely be used at the onset, especially when the agent is a powerful physiologically active substance with known properties. Successful treatment often indicates the pathological mechanism, and the considerable evidence that the *initial* lesions of the disease are caused by fat globule micro-embolism has recently been published.[4] This resulted from a study of decompression sickness affecting the nervous system, where gas bubbles can produce multiple sclerotic plaques in the spinal cord. Fat is the only other material known to reproduce the white matter plaques of multiple sclerosis in man and it is the only agent known to cause an acute and progressive leucoencephalopathy.

Unfortunately, the suggestion that fat embolism is the cause of MS has been interpreted by some as meaning that all the attacks patients suffer are due to embolism. It is only suggested that fat embolism is responsible for the initial damage to blood vessels at the onset of a new symptom.

Evidence of vascular damage has even been found to precede the onset of symptoms by 12 hours,[5] but the existence of blood-brain barrier disturbance is massively documented in acute attacks and has answered the question of which comes first, the vascular disturbance or the demyelination, because the radiolucency develops after a delay of several weeks. It is suggested that this crucial barrier may not heal completely, leaving the area vulnerable to the many onslaughts it is designed to resist. Most subsequent attacks therefore represent a relapse of existing symptoms triggered by anything that stresses the blood-brain barrier, from a common cold to a hot bath.

The evidence of this blood-brain barrier disturbance provided by modern scanning aids simply confirms the careful necropsy studies undertaken by Broman nearly 40 years ago. The integrity of the blood-brain barrier is, of course, a function of the oxygen content of the perfusing blood. Lower the blood oxygen tension and barrier dysfunction leads to diapedesis of red cells and the classical petechial haemorrhages of MS must indicate hypoxia, the cause is irrelevant, the action to be taken is obvious. We surely do not need to validate further the efficacy of oxygen.

Enlisting the aid of the latest and most exciting developments in scanning, NMR imaging has allowed the effect of hyperbaric oxygen to be illustrated in a patient with chronic MS. A scan immediately before and after a 90-minute hyperbaric oxygen session at twice atmospheric pressure has shown vasodilation in a periventricular plaque. A further scan which followed a course of 20 further sessions after a delay of three weeks shows the margins of the lesions are more circumscribed. The treatment was associated with considerable subjective benefit to the patient.

BLADDER FUNCTION

Commenting on these very preliminary results, Schumacher[6] has revealed that neurological expectations in MS are based at an unrealistic level. "To nail down the case for hyperbaric oxygen therapy," we would "have to show a reduction in the number or size of lesions in a controlled study."

Waiting the five years necessary to complete further double-blind trials to offer some amelioration of symptoms in patients with an established incurable disease seems, in view of the evidence, inhumane. Every study has confirmed improvement in bladder function and this has been carefully measured and documented. Bladder problems cause great distress to patients and are such a major cause of morbidity and mortality in the disease that this alone would justify widespread introduction of the therapy. Fortunately, the charity Action for Research into Multiple Sclerosis agrees with all these points and is establishing hyperbaric centres for long-term studies. Already six centres are operating in the UK.

REFERENCES

1. Fischer BH, Marks M, Reich T. N *Eng J Med.* 1983; 308: 181.

2 Chatak NR, Hirano A, Lijtmaer H, Zimmerman HM. *Arch Neurol*. 1974; 30: 484

3 Poser CM *Arch Neurol*. 1980; 37: 471

4 James PB. *Lancet*. 1982; ii: 380

5 Sears ES, Tindall RSA, Zarnow H. *Arch Neurol*. 1978; 35: 426

6 Schumacher GA. Reported in *Med Tribune*. 1983; 24: 4

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AN OPEN LETTER TO ALL BAROMEDICAL PHYSICIANS

Richard A Neubauer

I am concerned about the current worldwide explosion in the treatment of multiple sclerosis (MS) with hyperbaric oxygen (HBO). My concern is specifically about certain of the protocols being used for treatment.

At Ocean Medical Centre in Florida, we began our work with HBO for MS in 1973. The first publication of our studies was in 1978.[1] The original data provoked enough interest to lead to the funding of two animal studies.[2,3] A well-documented human trial was later performed by Fischer et al at New York University.[4]

In addition to these controlled research studies, there have been clinical studies involving up to 2,000 patients worldwide, to date. Following the publication of the results from the first 250 MS patients at Ocean Medical Centre,[5] I presented a report on the similarity of results between 500 MS patients and 100 MS patients treated in Italy, at the 5th Congresso Nazionale della Societa Italiana di Medicini Subacquea e Iperbaric in October 1982.[6] At the 8th Annual Conference on Clinical Applications of HBO in Anaheim, California in June 1983, I presented an international survey of reports on the HBO treatment of 1740 MS patients.[7] This was followed in September 1983 by an update and compilation of further controlled and/or longitudinal studies either underway or in the planning stages, presented at the First European Conference on Hyperbaric Medicine.[8] Many other reports have been published.[9,14,17,18]

One overwhelming fact stands out in these studies: All report encouraging results. Yet I receive several telephone calls and letters each week from MS patients regarding the deterioration they are experiencing with HBO. Rarely do I hear from their physicians.

Why is this? Especially in view of the extensive positive published reports. I believe that it is because these patients are being treated at a fixed pressure of 2 ATA (and occasionally higher), usually in a monoplace chamber.

For some reason, the fixed pressure protocol has been adopted at most centres which have started to treat MS with HBO since Fischer's publication. There is no concern about treatment differences between monoplace or multi-station chambers. The differences in the effect on the PaO_2 should be obvious to any baromedical physician (eg. Fischer's PaO_2 levels varied widely even with a fixed 2 ATA protocol in this multistation chamber). In the monoplace chamber, the PaO_2 is directly related to the pressure being used.

There is a scientific basis for the use of a variable, low-pressure protocol. My development of this approach was not entirely empirical. Research by reputable scientists, including Holbach, Wassman et al[14] and Kelly et al[15] clearly indicates that low and variable pressures are preferable in chronic neurological diseases.

This variable, low-pressure protocol has been widely used both in research and in clinical treatment. At Ocean Medical Centre we have treated over 700 MS patients with it. None have deteriorated due to pressure. The work of Fischer, et al[4] also lends support to this protocol. Their results showed that better clinical improvement occurred in patients having PaO_2 levels equalling those in a monoplace chamber at 1.4 - 1.6 ATA. Careful reading of that report would lead any physician to adjust the 2 ATA protocol downward, especially when using a monoplace chamber.

Additionally, the article recently published by Golovkin[16] in the USSR showed that MS patients exposed to pressures over 2 ATA for 20 minutes deteriorated rapidly. He now treats at 1.7 ATA in a multi-station chamber. Similar experience by Pallotta[17] and others in Italy led to the adoption of reduced depths. Davidson and James in Scotland,[11] using a multi-station chamber, changed from the Fischer protocol of fixed pressures to a lower beginning pressure protocol with improved results. Pressure is particularly critical to MS patients with abnormal nervous tissue, especially when optic neuritis is present.

There are three types of MS patients:

1) Newly diagnosed with early symptomology.
2) Stable chronic progressive.
3) Chronic progressive in exacerbation (relapsing/ remitting).

The variable pressure protocol starting at 1.5 ATA and ranging to 2 ATA is well established as effective in stable chronic progressive MS patients (type 2). Most of the published data on MS and HBO deals with type 2 patients. Invariably, HBO treatment leads to encouraging results when appropriate follow-up HBO treatments are given. Long-term longitudinal studies indicate that these patients secure alteration of the course of the disease.

Fewer early cases have been treated with HBO. They invariably respond, as they do with any other modality that is used. Further study is needed in this area. Such patients would have to have a longer follow-up period and more patients would be needed to differentiate between actual alteration of the disease and a placebo effect. James' comparison of decompression illness and MS led to his conclusion that all newly diagnosed MS patients should have HBO with the same priority as in decompression illness. (See his article in "Pressure Points", 13(5): 7-8, 1983 which appears on page 16).

The patient having chronic progressive MS with acute exacerbation presents a less clear picture than the above. My results in the treatment of such patients has not been as rewarding as those of McGehee in Houston, Texas or Pallotta in Italy. Recently James courageously treated a chronic progressive patient with an acute exacerbation including optic neuritis in a multi-station chamber using pressures up to 2.75 ATA. The pressure was cautiously titrated upward only after failure at a lower pressure. A dramatic result ensued. This experience warrants further cautious study. It does not indicate that all MS patients should be treated with such high pressures; deterioration is frequently seen in stable chronic progressive patients at 2 ATA or higher. This stage of the disease may require an entirely different protocol.

Understandably, research scientists find it difficult to use a variable pressure protocol in controlled studies. For them, I would suggest that better results might be attained with a steady pressure of 1.5 ATA throughout, rather than 2 ATA or higher, with particular reference to the monoplace chamber. In the monoplace chamber PaO_2 levels are identical with treatment at any given pressure. Only in the multi-station chambers used for research are PaO_2 measurements desirable.

There is one final concern related to the length of the initial series and follow-up treatments. The original Ocean Medical Centre protocol for MS called for 10 treatments in the initial series. This was raised to 20 when we observed that in patients who had longer initial series, results often did not appear until near 20 treatments. As many as 80 treatments have been given in the initial series in refractory patients.

Regression is predictable after the initial series when appropriate follow-up exposures are not given. It is remarkable that in Fischer's oxygen patients, who had only the initial series of 20 treatments, statistically less deterioration was noted at the end of a year. It seems unreasonable to me to withhold this treatment from research subjects. This is also not the appropriate way to utilize the published protocol.

As George Schumacher MD, noted in May of 1979 at the University of Vermont: In the treatment of multiple sclerosis "the only dependable evidence of beneficial therapeutic effect is stabilization, that is, no further worsening in the clinical status thenceforward .... Longitudinal comparisons over time of each patient's pre- and post-treatment status would provide the essential data". Results to date using the low, variable pressure HBO protocol are promising.

REFERENCES

1 Neubauer RA. Treatment of multiple sclerosis with monoplace HBO. J Fla *Med Assn*. 1978; 65: 101

2 Warren J et al. Oxygen immunosuppression: modification of experimental allergic encephalomyelitis in rodents *J Immunol*. 1978; 121: 315-320

3 Prockup LD, Grasso RJ. Ameliorating effects of HBO on experimental allergic encephalomyelitis. *Brain Res.* 1977: 221-225

4 Fischer BH et al. HBO treatment of MS a randomized placebo-controlled double-blind study. *N Eng J Med*. 1983; 308: 181-2

5 Neubauer RA. Exposure of MS patients to HBO 15 to 2 ATA. *J Fla Med Assn*. 1980; 67: 498-504

6 Neubauer RA. Keynote Address HBO in neurologic disorders. V Congr Naz Soc Ital Med Subac Iperbar. *III Ruin Com Mediter Med Subac Iperbar*. *Montecatini Terme*, 21-24 0ctobre 1982

7 Neubauer RA. Hyperbaric oxygen as treatment for multiple sclerosis. A comparison of 3 groups totalling 600 patients. *Seventh Ann Conf Clin Appl HBO*. Anaheim CA; June 1982

8 Neubauer RA. A summary of worldwide experience in the treatment of multiple sclerosis with hyperbaric oxygen. In: *(Program and Abstracts) First Europ Conf Hyperbar Med*. 7-9 September 1983. Amsterdam, The Netherlands; 36

9 Boschetty V, Cernoch J. Use of HBO in various neurologic diseases (preliminary report). *Bratisi Lek Listy*. 1970; 52: 298-301

10 Baixe JH. Bilan de onze anees d'activite en medicine hyperbare. *Medicine aeronautiaque et spatiale Medicine subaquatique et hyperbare*. 1978; Tome XIVV: n65

11 Zannini et al. 40 cases of MS treated with HBO therapy. *Min Med*. 1982; 3(42): 293-945

12 Editorial. Treatment of Multiple Sclerosis. *Lancet*. 1983; 308: 181-6

13 James PB. Oxygen for MS (Letter). *Lancet*. 1983; 1: 1161-62

14 Hammond ES, Ibarra EL. Evoked potentials after HBO treatment of MS *N Eng J Med.* 1983; 309(4): 241-242

15 Holbach KH, Wassmann H, Caroli E. Cerebral energy metabolism in patients with brain lesions at normo and hyperbaric oxygen pressures *J Neurol*. 1977; 217: 1730

16 Kelly KL, Lassiter K, Vongsuivut A et al. Effects of HBO and tissue oxygen studies in experimental paraplegia. *J Neurol*. 1972; 36: 425-429

18 Golovkin VI et al. HBO as immunity stimulator in disseminated sclerosis. *Sovetskay Med*. 1982; 12: 70-75

19 Pallotta R. Hyperbaric therapy of multiple sclerosis. *Min Med*. 1982; 73(42): 2947-2954

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ANZICS
ROCKHAMPTON MEETING OCTOBER 1983

We are grateful to the Queensland Regional Committee of the Australian and New Zealand Intensive Care Society for permission to publish papers which were presented at their inaugural annual meeting here in Rockhampton. The guest speakers were Dr Struan K Sutherland of the Commonwealth Serum Laboratories and Dr John Knight of SPUMS. Transcripts of the papers given at the second session on envenomation and the second session on underwater medicine appear below.

MANAGEMENT OF SPIDER BITE

Struan K Sutherland

I want to talk briefly about three spiders, the Red-back spider, Sydney Funnel-web spider and our Mystery spider, and to finally mention the Paralysis tick.

There are at least 2000 named species of spiders in Australia and perhaps 1000 unnamed. They all have poison glands except some of the little humped spiders. Even the Daddy-long-legs spiders have venom glands. Spiders are the most widely distributed venomous creatures in Australia and they show enormous variety. They are also one type of venomous creature that is found both inside houses and outside. This increases the opportunity for bites and stings.

RED-BACK SPIDER

The commonest reason for giving antivenom in Australia is the Red-back spider. This spider is found the length and breadth of this country and it is not just limited to outside toilets and back sheds. It is very common in the bush. Most people are bitten when they bring the spider into contact with their skin. This is a passive action such as when old clothing is picked up or gloves are used for the first time that day. It is the female which causes the harm, the male having fangs that are too small to penetrate human skin. It is closely related to the Black Widow spider in America. In most countries there are representatives of this spider which produce the syndrome called latrodectism. Per head of population we seem to get more cases of latrodectism than any other country in the world. Some countries like Italy have a little epidemic of the spiders every 10 years whereas we have it as a perennial problem.

The main toxin is alphalatrotoxin and it specifically acts at nerve endings. It releases transmitter substance and changes to nerves can be seen with the electron microscope. At the motor end plate this loss of transmitter substance produces a patchy sort of paralysis but most of the signs and symptoms are due to the effects on the autonomic nervous system where it releases catecholamines, to produce the classic syndrome. One can be bitten on the left hand and after a while there will be quite severe pains perhaps in the left foot and the right shoulder and arm will sweat profusely and then after a few more hours things will shift around. It is a strange disease.

First-Aid

In fact you really do not need any first-aid. The bite is moderately painful, it is like a mosquito sting at first but it then becomes quite painful over an hour or so. The venom works very slowly, so we do not recommend pressure immobilisation, you just take the spider and yourself safely to hospital.

Red-back Spider Antivenom

The antivenom has been available since 1956 and no-one has died since it became available. It is a very small volume antivenom and very rarely are there any reactions to it. Perhaps I should have mentioned this earlier but we do not believe in skin testing for any antivenom for sensitivity. It is quite unreliable and it wastes time.

THE SYDNEY FUNNEL-WEB SPIDER

A more interesting spider in some ways is the Funnel-web spider. It is unique to Australia and is the potentially most dangerous spider in the world. It is the only one which, for example, killed children in less than 90 minutes. Although bites and fatalities are rare, some three million people are at risk in the area around Sydney. The numbers of spiders are apparently increasing as people put in swimming pools and barbeques which produce more of the damp earth areas that the female spider likes. The male is the highly dangerous one. Without being sexist, this is the reverse of the normal situation in which the female spider is the more poisonous.

There are two very special features about the venom. One is that the venom affects mainly man and primates. The funnel-web venom will not kill rabbits, normal laboratory animals, mice, cats, dogs and so on. The other feature is that the venom has a specific action. Basically it attacks the outer covering of the nerves and causes spontaneous action potentials. It also disrupts some of the normal monitoring impulses coming down the nerve. The venom acts quite quickly and apart from hitting motor nerves, it attacks the autonomic nervous system releasing transmitter substance in a much more extensive fashion than with Red-back venom.

If someone is envenomed then within a few minutes they will get central effects such as nausea and headache. Muscle twitching can be extremely grotesque because everywhere the motor end-plates are firing off transmitter substance. Blood pressure can rise very dramatically perhaps up to 250 mm Hg systolic. The pulse rate of children can go over 200. Most patients develop generalised sweating.

Strangely enough it was not until 2 years ago that we finally determined why patients died. Dr Alan Duncan and Dr Jim Tibballs at the Royal Children's Hospital in Melbourne did a lot of work with CSL on monitored monkeys. The most important thing found was that sometimes when a monkey had received venom there would be a dramatic rise in the intracranial pressure which disturbed cerebral perfusion. It had the occasional effect of producing neurogenic pulmonary oedema so a monkey could have both impending brain death and pulmonary oedema. After looking back over the case histories we believe this is how many patients died. The unaided

clinician cannot detect sudden fluctuations in intracranial pressure. During this work Dr Duncan and Dr Tibballs helped greatly in the testing of the antivenom.

Funnel-web Spider Antivenom

The antivenom is different from any other type of antivenom in the world, being of very small dosage and consisting of a pure rabbit immunoglobulin. It works very quickly because it is an intact immunoglobulin. Fortunately the toxin that it is going to attack is mostly still on the outside of nerves so it is easily reached. Experimentally one can see a reverse occurring within 2 or 3 minutes.

The first time the Funnel-web antivenom was used was for a 49 year old man who was transferred from the Ryde Hospital to the Royal North Shore some 90 minutes after he had been bitten by a Funnel-web. When admitted he was comatose with pupils widely dilated. They had great trouble controlling him because he was extremely irrational and tore out his IV line and so on. On admission his blood pressure was over 200 and his pulse rate was 160. He had 2 doses of antivenom and within about 30 minutes or so his blood pressure was down to normal and he had regained consciousness. Dr Malcolm Fisher reported that half an hour later monitoring was no longer required and the patient was sitting up in bed waiting for his dentures to come in so he could go on television. This shows the speed at which the specific biological antidote can work in a situation where the toxin is easily reached. The antivenom has been used a number of times now.

There is another Funnel-web which we have kept fairly quiet about which is found all the way up from Central NSW right up north to Toowoomba and it is probably more dangerous than the Sydney Funnel-web. It is the tree dwelling *Atrax formidabilis* which is much bigger and the female is highly poisonous. Bites by this spider are very rare because it inhabits areas where there are few people. Fortunately, the antivenom made against the Sydney Funnel-web spider neutralises this spider's venom.

A MYSTERY SPIDER

There is a new disease which we really first became aware of some 5 or 6 years ago when a housewife suffered a strange injury. She lived in a farm house in Northern Victoria. She went out mid-morning one day and planted a couple of bulbs immediately outside her back door. That was the only time she left the house that whole morning and the actual planting only took a few seconds. About half an hour after she had planted the bulbs her hand became painful. Over the next 24 hours she developed a critical illness with severe full thickness damage to the skin of the hand and a systemic illness involving diarrhoea, vomiting and extreme toxicity. There was a total lack of positive laboratory findings. A marked feature of this case was the severe pain. She had had her second baby four weeks beforehand and found the pain of this episode something like ten times worse and wanted her hand cut off.

Since then, we have collected perhaps 35-40 cases of various degrees of injury in which often the bite is not noticed or is considered very minor. They have then progressed to either just superficial injury which resolved or full thickness skin loss.

One severe case involved a little girl of two and a half. She went into a storeroom with her father just as the school was opening up in February in Melbourne. She came out and sat down looking at the top of her foot, she just had a thong on and said something had bitten her. After a while she ignored it and ran round quite happily for a few hours but then became ill. Over the next 24 hours she developed a wide area of cyanotic skin that went on to break down to full thickness necrosis. At the same time she developed a systemic illness which was equated to cholera. She required something like a quarter of her circulating volume to be replaced as an emergency

I will summarise what we have gathered from these cases.

They have occurred in all eastern states of Australia. The problem is that the creature involved is usually not found. This is quite different to bee sting and snake bite and so on where one generally at least sees the culprit. There are variable changes that occur at the bite site. Possibly there are three or four different types of spiders that are producing these effects. It might be the spider is sometimes injecting a lot of venom and at other times not.

Some cases develop quite marked local pain and a sterile cellulitis. It may be very very painful for 24 hours and then the area returns to normal. Sometimes small punched out neighbouring lesions are seen as though the creature crawled along and made a couple of bites. Gardeners may have such injuries by Thursday if they have been gardening during the weekend without gloves.

Sometimes there are horrifying local changes with gross oedema. When the surgeon removes the dead tissue it looks as though a cytotoxic drug has been injected subcutaneously, because the fat is liquified and it just pours out. It is these severe cases which are associated with the general illness. There is no temperature initially, they might be in fact hypothermic. They can be quite demented with pain. There may be shock and quite prolific diarrhoea. The laboratory investigations really have not helped us other than to exclude things. The biochemistry and coagulation profiles are basically normal. Sometimes the white cell counts are elevated and there is a toxic suppression of neutrophils change but nothing that is particularly special. In some cases we have found the IgM has been elevated but that has not been consistent. All cultures of blood and lesions have grown nothing to date and the histology of the ulcer margins has been unproductive.

The most likely culprit at the moment is the White Spotted spider *Lampona cylindrata* which is very common right around Australia. It is a grubby looking little spider which drags its cylindrical tummy along. Often it is found in bathrooms or bedrooms. We have got definite cases of bites by this spider that have broken down and caused marked ulceration. That is the end of the story to date.

We are just waiting for more cases as we have to have the proven culprit to work on and then get support to work on an antivenom. In the meantime, as the cases come along we cannot do anything about them apart from photograph them.

AUSTRALIAN PARALYSIS TICK

Just for completeness since we are talking about spiders and arachnids, mention should be made of the Australian Paralysis tick which is found in a wide area from Mallacoota to Cairns. The adult form is particularly dangerous because as it is engorging and burying itself temporarily in the skin of a human it may release a very potent neurotoxin. It is an unusual neurotoxin because it works extremely slowly. One can inject it into a dog and nothing will happen for 18 hours and then the dog will start getting paralysed. This toxin acts presynaptically and reverses quite slowly. First-aid is to gently remove the intact tick. There is an antitoxin but it is one type of paralysis which recovers, in most cases, with just standard intensive care. Once the tick is removed, the victim generally improves quite rapidly.

THE BOX JELLY FISH STING

John Williamson

By any accounts *Chironex Fleckeri* is a significant animal and the number of fatalities that it has produced will support that statement. The Northern Australian and Western Indo-Pacific box jellyfish has now been responsible for 68 documented human deaths in this country, 70% of whom were women and children, due to their generally smaller body mass and hairless skin. The whole crux of the problem of the box jelly fish is related to the speed with which this animal can produce envenomation in human beings and of course in its natural prey. It has already been pointed out today by Dr Sutherland in his opening address that animals including this one have no interest whatever in human beings. All envenomations by this animal are due to accidental encounters and it is the human's fault. The animal does not attack. It is pertinent to say that none of these fatalities have received any effective form of resuscitation to date.

When a snake envenomates, or a spider or a blue-ringed octopus or a cone shell, the venom is all deposited in one place so there is a limited surface area, although obviously an effective one, between the venom parcels and the blood bearing tissues and the lymph bearing tissues where the absorption occurs. However, with the box jelly fish, the venom is divided into many thousands of millions of tiny parcels spread over the architecture of the tentacles. The nematocysts occur on transverse bar-like patterns on the tentacles. Consequently, when envenomation occurs the venom bearing nematocysts or microbasic mastigophores, as our colleague Bob Hartwick likes to call them, discharge their venom in multiple million tiny doses and inject them into the victim in many different sites at the same time. The trajectory of most of these nematocysts is something just under a millimetre which will carry the venom into the subepidermal, richly vascularised tissues. This arrangement of envenomation offers an enormous area for absorption so the speed at which high blood levels of toxin are achieved following a serious envenomation by one of these animals is extremely rapid. It is measured in minutes.

Small victims die very rapidly on the beach. A large number of the fatalities and the non-fatal stings involve the Aboriginal population in this country.

In a survivor of a serious sting, there is a much more vigorous inflammatory response. One can see the actual cross hatched or ladder pattern which is diagnostic of the sting of this jelly fish. The only other jelly fish that may produce a pattern like this, and it will not be as dramatic, is the fire jelly.

Three to ten per cent acetic acid in water (vinegar), as far as our studies have shown, renders the nematocysts of the box jelly fish irreversibly inactive within a period of about 30 seconds. Nothing we have been able to do can provoke them to fire following exposure to that concentration of acetic acid. Methylated spirits causes a massive discharge of nematocysts from the tentacle of the box jelly fish. If that tentacle happens to be applied to a human victim, then there will be increased envenomation. When the tentacle has been treated with vinegar (4-6% acetic acid), it does not matter whether it is brown vinegar or white vinegar, and then methylated spirits is applied there is no response. Time prevents me from discussing the role of vinegar in the treatment of other jelly fish stings.

I must emphasize that it is important to understand that vinegar does nothing for the pain. It inactivates the unfired nematocysts but it has no effective role to play whatever in the treatment of the pain of the sting of the venom that has already been injected. Vinegar does work for other jelly fish but certainly not for all. Vinegar certainly renders the nematocysts quite harmless and useless.

We advocate, purely by extrapolation and without any experimental proof at this stage, the use of compressive immobilisation. We would advocate in any serious sting, and certainly any sting where resuscitation becomes necessary, the immediate application of vinegar followed by the application of a compressive immobilisation bandage over as much of a sting area as possible. Remember compressive bandages are not tourniquets. This treatment appears reasonable to us at this stage while we await experimental confirmation that this will trap the venom in the skin. However, remember this has to be done immediately, because the speed of absorption of this venom is such that unless it is done immediately it is unlikely to have any greatly beneficial effect.

Critics of this approach, in the absence of experimental verifications could well say that one is not doing the best for the patient because this will trap the venom in the skin and will produce increased pain. The answer is, I feel, that priorities are important. If the victim is unconscious increased pain will not matter. If he or she has been seriously stung, it is better to have severe pain than to run the risk of losing ones life. The dermato-necrotic, or skin killing, effect will have, theoretically at least, been made worse by this action. There is no doubt about the powerful skin killing effects of the venom. However where life is threatened, this may be the lesser of two evils. The circumstantial evidence that we now have, which is extensive and strong, shows that the administration of antivenom will reduce both the pain and the skin killing effects. Obviously resuscitation takes absolute priority. Our recommended emergency treatment on the beach now consists of vinegar dousing, "pressure-immobilisation" of the sting area, and application of ice-water through the compressive bandages for partial pain relief in the conscious

victim and the early intravenous or intramuscular administration of specific antivenom (concentrated Chiron*ex Fleckeri* venom-specific immunoglobulins, isolated from the serum of hyperimmunised sheep) with appropriate precautions. That is the sequence of events that we advocate, particularly when we are teaching the surf life savers who patrol beaches in the risk area and other members of the public.

When talking to a group of intensivists, this problem is a little academic because everything happens so quickly that it all happens on the beach and by the time the victim, if he is alive, gets to the intensive care unit, it is largely all over bar the shouting. Unless the intensivist happens to be on the beach or in the ambulance he is unlikely to see any of the actual shooting. That is not to say that a knowledge of this is not important for intensivists. When the patients reach intensive care follow up treatment makes a big difference. But nothing takes the place of immediate and effective treatment on the spot. Before I leave this subject of resuscitation, let me re-emphasize what has already been emphasized today, that when a person is being resuscitated from a massive envenomation, when resuscitation is effective absorption of venom will occur again and unless it is trapped in the skin or neutralised by antivenom one can expect a further collapse from systemic effects. So one has to keep on working and one does not leave the patient.

The characterisation of the venom is incomplete at this stage. It has two broad molecular weight groups. The study of the lethal factor which includes the so-called cardiotoxic factor has been done on guinea pigs and rabbits, mice, rats and toads. It worries me a little bit to extrapolate of some of these conclusions to the live human clinical situation on the beach.

There are a number of interesting new developments in this area. For example, recently in the Medical Journal of Australia some workers from Maryland published encouraging results in mice where verapramil reversed the arrhythmic potential of *Chironex Fleckeri* venom. They suggested that this may be an approach to the first-aid situation. We have only two well documented severe envenomations that have been survived. They both received effective resuscitation on the beach. But in both those victims, one a child and the other a pregnant adult female, only expired air resuscitation was necessary. There is no doubt that they received a potentially lethal dose of venom. The question I ask is whether the danger to the adult human or the child human is a cardiotoxic one from this venom or is it something that is acting centrally neurologically which produces respiratory arrest. I am sure that the venom does not do the myocardium any good at all, but it may not be the critical factor. It would appear on the evidence so far that expired air resuscitation alone, if effective and sustained, will be all that is required in the absence of some other complicating factor. The haemolytic component appears to be clinically unimportant. Why does apnoea occur when the venom is extremely thermolabile? When it is raised from the temperature of the sea to the temperature of the human body its longevity may not be very great. Effective resuscitation for those two cases was only really necessary for a period of about 30 minutes to three quarters of an hour before spontaneous ventilation recurred. This fact has important implications in teaching first aid and in the approach to the problem.

The administration of the antivenom is quite another problem. There is no question that it is effective, at least on the basis of the cases that we have recorded so far. Vic Callanan, Max McDonald and I, among others, are teaching the life savers in the risk part of the world, to give antivenom intramuscularly to people who obviously require it. It is not difficult to decide when a case does require antivenom. The antivenom is a concentrated mixture of immunoglobulins from hyperimmunised sheep so it carries the risk of serum reaction and all appropriate precautions should be taken which have been discussed this morning. When the choice is between antihistamines, adrenalin and steroids it is not hard to understand why we have recommended that the life savers give steroids and not one of the others on the beach. The whole subject of box jelly fish envenomation is heavily influenced by the need to be practical. It is no good telling life savers to give adrenalin and it is not much better to ask them to give antihistamines to someone whose conscious state may already be impaired. So it seems to us that steroids and antivenom is the best choice on the beach until the patient gets to an area where more expert medical treatment is available. The antivenom is supplied in ampoules, from the CSL. We recommend three ampoules intramuscularly on the beach assuming that the people there cannot be expected to give an intravenous injection. If intravenous access can be obtained that is excellent. Obviously that is the route of choice but it is most unlikely to occur on the beach. If you have ever tried to do a venipuncture on a wet, sand covered limb in a shocked or struggling patient, you will know that it is just about impossible. As for one in ten dilution on the beach, that is impossible too. Intravenous antivenom is certainly desirable in the casualty or the intensive care unit.

A lot of claims have been made about pain relief. We have looked at a lot of substances applied to box jellyfish stings and *physalia* (blue-bottle) stings. None of them seem to do anything whatever for pain relief, or for anything else such as nematocyst inhibition. The only substance which offered any sort of pain relief was Skefron which is a volatile complicated hydrocarbon which appeared to achieve its effect by simple cooling. It seems to us that from a practical point of view on the beach in the risk period in the Northern part of Australia and for that matter, elsewhere in the world where the risk occurs, if the person is conscious and in a lot of pain one might try reducing the pain by applying ice-water or ice directly over the sting area through the bandages. Ice is likely to be available on the beach in summer at a barbecue. Certainly they will not have a complicated hydrocarbon handy and Skefron costs a certain amount of money. It is a very small can and I have no idea of the potential toxic effects of that agent. The message is, at our present meagre level of knowledge, to be extremely optimistic and extremely aggressive and teach the lay public, because they are the people who are going to be on the spot when this problem occurs, the proper first aid and the need for protective clothing.

There are three jelly fish stings and one other toxic marine animal sting that have either been published or come to my notice, which resulted in detectable antibodies and produced a subsequent reaction some weeks later in the absence of

further tentacle contact. One is the sea nettle which in America is a Chr*ystosora* animal, the closest relative we have in Australia is *Pelagia* or the mauve stinger. Another is their Man O' War which is very similar to, if not identical with, our Portuguese Man O'War or blue-bottle *(physalia)*. Published work shows that antibodies, particularly IGG but also IGE antibodies, have been measured in people stung by these animals and the antibody titre is correlated with the severity of the sting and of the symptoms. The other two animals are the box jelly fish, and a toxic sponge which came into contact with the hand of a diving Adelaide surgeon. These two are cases I have seen or been consulted about. The antibodies of at least the first two appear immediately. They may persist for years. In the case of the girl who was stung by a box jellyfish at Yeppoon about 16 days later the whole thing blew up again. It was clear that this was not infection. In fact this secondary reaction caused more problems for her than did the primary sting. She did have a history of allergy. When I was contacted on the phone I advised that she be treated with steroids, as I had done with the chap in Adelaide, and she got better.

It looks very much as if allergic reactions in jelly fish envenomation may be important. This applies to the immediate reaction as well as to delayed reactions. Elevated specific immunoglobulins, particularly IGG and IGE, have been demonstrated particularly with the sea nettle and *physalia,* and these can persist for years. Recurrence of clinical cutaneous reaction to jellyfish stings may occur within a few weeks without additional contact with the tentacles. As far as sea nettle and blue-bottle are concerned serelogical cross reactivity occurs.

SEA SNAKE ENVENOMATION

Hilary Mercer

My presentation concerns a case of a sea snake bite which is apparently the first case which has been reported in the Australian literature, although there have been many cases reported from Malaysian waters.

A couple of years ago a two year old child was paddling on Lamamoor beach which is quite a picturesque spot between Emu Park and Yeppoon. She started screaming and the mother ran down and saw a rather loathsome creature attached to the child's ankle. As she approached the creature swam away towards two teenage boys who killed it and brought it along for identification. The mother had great presence of mind and grabbed her daughter around the calf with both her hands and did not let go. The pair of them were taken to the Yeppoon Ambulance station where the wound was washed and inspected by the ambulancemen. No tourniquet or compression bandage was applied.

The mother removed her hands. Up to that point the child was speaking coherently and quite bright. But within 30 seconds of the mother taking off her hands, the child became very weak, developed ptosis and some respiratory distress. They were rushed to the Yeppoon Hospital, which was close by, where about 20 minutes after envenomation the child became cyanosed and needed intubation. From there they went to Rockhampton Hospital. They arrived there about an hour after envenomation. By this time the child tolerated reintubation without any resistance whatsoever.

Then about one and a half hours after envenomation, we gave the first dose of sea-snake antivenom. By this time the snake had been brought along and identified by one of our local herpetologists, and this was later confirmed by the Queensland Museum, as being *Astocius Stoksii*. Incidentally, no antihistamine was given because of a previous reaction to promethazine and for some obscure reason adrenalin was not given either.

Over the next two hours there was no real improvement and we gave two further ampoules of antivenom. After the third ampoule there was some apparent clinical improvement. The child opened her eyes and started looking about. However over the next 10 hours or so the child seemed to regress and 14 hours after envenomation the child had odd clonic movements and we thought the conscious state was deteriorating again. We treated her with phenytoin and gave a fourth ampoule of antivenom.

I then spoke to Struan Sutherland on the phone and he suggested that we were probably not giving enough antivenom. So we gave another three ampoules. The only thing that stopped us giving more was that the child developed a rash which responded rapidly to antihistamines. After those further three ampoules the child became a lot better. About 22 hours after the bite we were able to extubate her. Two hours after that she was sitting up and attempting to speak.

The following day she was sent to the children's ward. Over the next few days she had very odd movements of her limbs and hallucinated but she was able to be discharged six days after the envenomation. Subsequently there were no real problems. However she must have had sadistic brothers because they kept creeping up to her with bits of grass and things and saying "Ah, the snake's got you!" and she would go all 'funny'.

There was nothing dramatic about the investigations. The coagulation status was normal, muscle enzymes were up, but cardiac enzymes were normal. The white blood cell count was raised, as expected, to about 27,000. Renal function tests were quite normal. Myoglobinuria was only found on one occasion about 48 hours after the envenomation.

The snake itself was about one and a half metres long. It was an *Astocius Stoksii* which is a snake that is not seen very often around here. It inhabits the waters of the Indo-Malayan coast more than here. We see many sea-snakes in central Queensland. They are regarded as potentially dangerous by divers and fishermen but we do not see many bites. They seem to be timid creatures. However when they are mating they conglomerate in large number and may come towards people which is very unnerving apparently.

The way this child's foot was mauled may have been responsible for the massive envenomation in this case. This snake is the largest of the sea snakes. It has the largest mouth with the largest fangs, as far as I know, of the sea snakes. Its fangs can penetrate wet suits.

There are just a few points worth making. The bite itself is not painful. We were lucky that the creature was seen and caught, otherwise I do not know what we would have thought. A little child playing among the rocks, would we have treated it as a sea snake bite? Other things that one might consider, stonefish, bullrouts, box jellyfish, are characterised by intense pain whereas this was not. What was the value of first aid? Whether or not the mother's grip of the leg was very important, I do not know. Whether the sudden collapse of the child would have occurred when it did or not is unknown. But one imagines that had a compression bandage been put on at the ambulance station, the course may have been a little less dramatic. Finally, although this is quite a rare occurrence, there are more and more people involved in water sports and diving and so forth, so it could happen again. Certainly coastal communities around Northern Queensland should be aware of the possibility and have access to sea-snake antivenom.

ENVENOMATION BY THE BLUE RINGED OCTOPUS

Hugh Stephens

When I arrived from the UK to take up my appointment at the Gold Coast Hospital I had never heard of a Blue Ringed octopus. I took it to be a man-eating monster of Jules Verne proportions. There was obviously someone else of equal ignorance in Queensland at the time. He had picked up the thing, died and was resuscitated. He was born in Mauritius and had spent much of his life in New Zealand before moving to Sydney where he worked as a bus driver. Thus he had no particular exposure to a Blue Ringed octopus previously. He was a member of a group of holiday makers who went on a day trip to South Stradbroke Island. Returning to the launch that conveyed them to the island he picked up two small octopi from a pool to show them to his two nieces who were with him. When he had discarded the second octopus there was a drop of blood on the back of his left hand, although he had been unaware of any bite at the time. A few minutes later he was relating the incident to the skipper of the launch when he felt a degree of numbness and tingling around his mouth. This was followed by weakness of his legs which caused him to collapse.

Fortunately for him there were nearby an off duty customs officer and an off duty ambulance officer who were talking to a seaplane pilot who, luckily, was on duty and had his plane with him. The ambulance and customs officers were both efficient in cardio-pulmonary resuscitation and, in fact, had just attended a revision course. Also the ambulance officer had recently read a headline in the local Gold Coast paper which had alluded to a plague of these monsters hitting the Gold Coast. Consequently he had read up the symptoms and signs and treatment of the bite. In short, God was with the patient that day. The trio overheard the conversation, and with the skipper came to the patient's aid. The urgency of the problem was not lost on them. They immediately bundled the patient onto the plane, radioed for an ambulance to meet them at the other end and took off to the mainland a few minutes ride away.

Approximately three minutes later the patient was noticed to twitch mildly in the plane and lost consciousness. As he did not have any pulse or respirations CPR was commenced. On landing the patient was transferred to the ambulance where CPR was continued and oxygen via a resuscitator replaced the mouth to mouth. The patient was taken to the Gold Coast hospital where he arrived two or three minutes later.

Examination in the Accident and Emergency department showed him to have fixed dilated pupils, no eye opening, no motor or verbal response, no pulse, no respirations and asystole on the Lifepak monitor. He was resuscitated with intubation, ventilation, intravenous adrenalin, sodium bicarbonate, DC counter shock. Sinus tachycardia and a spontaneous cardiac output were restored. His problems at this stage were paralysis from the bite, aspiration pneumonitis and anoxic ischaemic encephalopathy and possibly brain death. His management consisted of hyperventilation, dexamethasone, intravenous mannitol and antibiotics in the form of Amoxyl. The major concern at this stage was our inability to distinguish between brain death and the effects of the venom. Fortunately the patient was obviously comatose because I gather that a lot of conversation went on between the residents as to whether he was dead or not dead. Four hours post admission the patient was noticed to have some reflex withdrawal from painful stimuli of both hands and feet. At five and a half hours post admission his pupils were mid range and reactive, he had cough and gag reflexes and spontaneous movements of all limbs. Eighteen hours after admission we were able to extubate him. His cerebral status was still giving us cause for concern as he bad no comprehensible conversation, although he had spontaneous eye opening and no focal neurological signs.

Over the next few days his mental status gradually improved. Between 24 and 48 hours confusion and disorientation gave way to a period of sexual harassment of the nursing staff. We were assured by his wife that this was definitely abnormal. He had no recollection of the events of the day throughout his stay of ten days. He was transferred to the Canterbury Hospital in Sydney on day eleven. By that stage a certain degree of confabulation had occurred and he claimed to have remembered the incident and wrestling with the octopus which he had said had a six foot span.

To summarize, a 44 year old male was bitten by a Blue Ringed octopus and within three minutes developed circumoral parathesiae, by four minutes limb weakness and collapse, and in seven minutes cardiorespiratory arrest. The complete paralysis persisted for up to about four hours. Reflex withdrawal from pain was then noted. Over the next four to eighteen hours he had gradual return of motor function, the course of which may have been modified by anoxic ischaemic encephalopathy.

Since then we have had another case of Blue Ringed octopus bite. A child found a Blue Ringed octopus in a coke tin and picked it up. He had actually kicked the tin first, and had noticed a mark on his foot. This was about a month after the first chap. The boy brought the octopus to casualty in his hand and pointed to the mark on his foot. So everybody was waiting for him to collapse. We sat there for 12 hours waiting for him to collapse but nothing

happened so we sent him home the next day. Perhaps his Blue Ringed octopus gave a "dry" bite.

STONEFISH, CONUS SHELLS AND BLUE RINGED OCTOPUS

Struan K Sutherland

THE STONEFISH

The Stonefish is found on nearly two-thirds of the Australian coast. It is the only stinging fish which has been known to kill people but there are no recorded deaths in Australia. The fish has 13 venomous spines but does not use its venom for collecting its food but more for protection. It is a very solidly built creature and if stood on the venomous spines may go deep into the sole of the foot. Not only is venom spurted in but actual parts of the venom gland enter and this causes extreme pain. As the fish is usually buried right up almost to its eyes in sand and coloured algae it is very very hard to see. Bob Endean says one can pick them up and put them down in their natural environment and spend half an hour trying to find them again. Some people say that a Stonefish is a bit like a politician. It sits round doing nothing all day, has a big mouth and is highly venomous if you try and shift it!

Severe local damage and extreme pain is produced by the Stonefish venom. Experimentally there is some evidence that it can effect cardiac muscle but I am not sure that this has been shown in humans.

Management of Stonefish Stings

We know that antivenom will quite dramatically reduce the pain and prevent necrosis but often one is in a situation where there is no immediate access to antivenom.

First-Aid

There is no place for restricting the movement of this venom because it is causing severe pain and tissue damage. The venom should be encouraged to move away to dilute itself, so do not apply pressure immobilisation. Never apply a tourniquet. Use warm water for pain relief of the injuries. Bathing the foot or hand in warm water increases the circulation of blood through the area. Some marine toxins are very heat labile and perhaps warm to hot water helps detoxify them. Getting hot water might be a problem but usually one can use cooling water from an outboard or inboard motor for this purpose. Often pethidine or morphine do not relieve the pain of a severe Stonefish sting. A severe case may need surgery to clean the injury up, so under such circumstances consider a regional nerve block with, say, bupivacaine to give the patient lengthy relief of pain. This will also allow debridement of the injury.

CONUS SHELLS

There are many Conus shells, three of which are known to be highly dangerous to man. One species, *Geographis*, is the most dangerous. Conus shells are more or less sea going snails that produce very toxic venom. They have developed tiny harpoons which are soaked in venom. When a little fish that they would like to eat swims past, this harpoon soaked in venom is moved up to the front of the mouth and pushed into the fish which quickly becomes paralysed. Then the creature can open its mouth and quietly cover the fish and eat it. The harpoons are exquisitely made. They are only about a centimetre long, and the barb is different in each species. They are hollow and are only used once and are designed for the type of prey that the Conus shell likes. When a human is stung the harpoon can penetrate quite deeply. The toxin is unique. The *Geographis* has a little polypeptide of ten amino acids which acts postsynaptically and acts very very quickly. There is no antivenom for that particular toxin but the suggestion is that it would wear off in time like the octopus toxin. For Conus shell it seems reasonable to use pressure immobilisation for first aid. The main thing of course is not to pick them up because they can bring their mouth parts and harpoon almost to the other end of their body.

THE BLUE RINGED OCTOPUS

There are two species, the northern one and the southern one. I get the impression the southern one is far more common than the one found in tropical waters. Fully grown it is about 5 inches long, and carries enough toxin to paralyse perhaps 10 men. As far as I know it only bites people if they pick it up and hold it against their skin and restrain it. We had one person bitten under water and that was when he saw an octopus go into a hole on a pier. He stuck his finger in and I think any octopus has got every right to bite someone who does that! It seems reasonable to say that underwater the octopus poses no threat at all to the sensible diver who leaves it alone.

The anatomy of the octopus is quite fascinating. Where the arms join there is a little parrot-like beak. Its oesophagus goes upwards between its eyes, through its brain, and its stomach sits up on top. That is why it is called a cephalopod, head to foot. Near the stomach are two kidney shaped salivary glands, which produce the toxic saliva. The saliva flows down a duct to be released through the mouth. The octopus normally uses the toxin to paralyse crabs. It has a very delicate skin so it does not like getting in and fighting. If it sees a nice juicy crab it can swim over and just spray some saliva around the crab. When the crab gets ataxic and partially paralysed the octopus settles down and eats it. It is only if humans pick it up and annoy it, that it will sink its beak into the human. It is interesting that it is mainly adults who have been bitten.

The toxin from the Blue Ringed octopus is in all probability tetrodotoxin which is of course found in Puffer fish and Toad fish. The same toxin is also found in the Californian newt and in certain South African frogs. Tetrodotoxin acts specifically on the sodium gates in nerves. By stopping the movement of sodium, it very promptly blocks the movement of action potentials and hence produces a flaccid paralysis within a few minutes. Tetrodotoxin has a molecular weight of 319, and it is heat stable.

Management of Bites

If possible, the pressure immobilisation type of first-aid should be applied to the bitten area. Paralysis will wear off and the patient fully recover if adequate artificial ventilation is promptly instituted. This may have to be maintained for some hours.

Finally, there are still many venomous creatures that should be investigated. There are many Australian venoms that we know nothing about, such as some of our ant venoms. I hope that if anyone gets a chance to encourage collaboration and local research into the venoms in his area, he (or she) will do so.

Question

Is topical local anaesthetic any good for relieving pain in sea wasp stings?

Dr John Williamson

In our experience, no. The pain is much too severe to respond to that sort of application. We did ask the College of Dermatologists what they thought about the routine application of lignocaine as a cutaneous treatment for stings and they reacted violently. They thought it was a bad idea because a percentage of the population is allergic to lignocaine which has toxic actions of its own. That is a very conservative approach. Anyway with box jelly fish stings lignocaine does not seem to help at all.

With other minor stings, it does seem to help. But we do know that in the treatment of any marine sting, particularly jelly fish stings, the placebo effect is quite profound. We believe that that is why methylated spirits held its own for so long. As long as somebody sees and feels that something is being done, they feel a bit better about it. This particularly applies naturally to the parents of stung children. There is no doubt that in the single blind studies we have done, the placebo effect of treating a sting is quite profound. That is a legitimate approach, because it is cheap and it does no harm, but you have got to exclude the placebo effect when you are evaluating treatment.

Question

Could Dr Sutherland give us his views on the potential toxicity of the Toowoomba Funnel-webb. During the summer months, this spider can be found in quite large numbers in the Toowoomba Range. In the last two years, I have managed three bites. On two occasions, the spider had to be actually prised off the finger or the toe that it had bitten. In none of these cases did the patient come to any harm. In fact, talking to colleagues who have been in Toowoomba for some time, I have not been able to find any documentation or medical records to suggest that anyone in the Toowoomba area has come to harm as a result of one of these bites.

Dr S Sutherland

Only one in ten people, or it might be one in five, that are bitten by a male Funnel-webb gets sick because it has usually lost its venom. We have done some work on some of the Toowoomba spiders that have been identified and classified. The venom seems to be particularly toxic and we know the Sydney Funnel-webb antivenom neutralizes it.

I do not wish it on the Toowoomba people but you will get a case of envenomation in time and that is why you hold the antivenom. You might get a case tomorrow, or you might not get one for five years. Incidentally no-one knows why the Funnel-webb venom is only effective against the primates.

DECOMPRESSION SICKNESS

CASE REPORTS

J Orton

I have two cases of decompression sickness to present. One was definitely decompression sickness and the other suspected. The famous American Catholic speaker Bishop Fulton Sheen once told his flock, “There is no pleasure without pain”. His theological comments were not actually directed at anything we are talking about today but they may well have been directed at sports diving.

The first case was a 29 year old, fit fellow who was PADI trained with no significant past history, who developed obvious neurological decompression sickness following repetitive diving. He had about 70 hours experience diving. He had been on a weekend diving trip where he had really pushed himself right to the limits of the tables and in addition there were a lot of other contributing factors. He did four dives on the Saturday, none over 60 feet, that is day one, and two dives on the Sunday, one to 60 feet and one to 80 feet. Both these dives were within the US Navy tables no decompression (no stops) limits, but they were only just within. He was obviously pushing it a bit. Using the Royal Navy system, he was beyond the limits of the tables for repetitive dives and he should have done a stop on the first day. Now consider the additional factors. There had been a lot of merry making the day before with a bit of alcohol involved. Significantly, he did not even go to bed the night before his first day’s diving. He was up all night, Friday night. He was taking Sudafed (pseudoephidrine) tablets for what he said was “blocked ears”. He had a very vigorous approach to the whole weekend’s diving. He was always in the water, always swimming and he did a lot of snorkelling between the dives. There was also a lot of vigorous activity while he was in the water doing his tank dives as well.

During the ascent on the last dive he became disco-ordinated. He got vertigo and felt quite weak. He got up to the surface and had a rest and improved a bit but still felt weak. However he was able to help stow the gear. He had some difficulty passing urine after that last dive. There was some hesitancy and dribbling. On the way back, he

kept on telling people that he was not well. But back on shore that Sunday night he did not give up. He went to a barbecue, had a few drinks, but finally went home to bed. On the Monday he woke up still feeling unwell and noticed his weakness was increasing. He was quite drowsy and had a headache. He was quite nauseated after breakfast but did not vomit. He went to his local doctor. There he was noted to be quite drowsy. He was unable to stand up. When asked to come into the doctor's room, he had to push himself up using his arms on his thighs, he did not have the strength in his lower limbs to stand. The examination on that day showed obvious weakness in all muscles and all limbs. He could not straight leg raise. But there were no sensation changes. So the diagnosis of neurological decompression sickness was made. It took a little while to organise recompression but that evening it was underway in the Australian Institute of Marine Science (AIMS) recompression chamber about 40 kilometres from Townsville. This is quite a good size chamber and reasonably good backup medical facilities.

During the time before recompression the patient was given oxygen to breathe from a mask. The recompression treatment consisted of an extended table 62 on 100 per cent oxygen. After just 5 minutes at 18 metres he said his head felt clearer, his breathing felt much easier and he had an obvious increase in muscle power, to the extent that he could straight leg raise straight away. All within five minutes so the diagnosis was clear. After an hour, he could sit up unaided. He had three hours at 18 metres then we came up to 9 metres for an hour. Then he was noted to be quite a bit weaker than previously and he did not feel as clear mentally. So we took him back down to 18 metres for another two hours. At this depth he had no symptoms. He had four hours at 9 metres with no further symptoms. When he got back to the surface he said he felt completely well.

Over the next 12-24 hours he had a slow return of symptoms particularly the weakness. It was decided to continue recompression treatments until no further demonstrable improvement was seen. So he had repeated hyperbaric oxygen treatment until there was no residual weakness and no residual deficit and quite clear mentation. He subsequently made quite an uneventful recovery with no deficit at all.

To summarize, a fit 29 year old man pushed himself right to the limits of the tables and thrashed himself physically all weekend. He suffered weakness and mental changes from neurological decompression sickness and was successfully treated with recompression. In this case symptoms were present on ascent. I think that it is important to make people realise that if that happens, something must be wrong. He told people on the way back that he did not feel well, yet no one in the party thought that he should see a doctor. He spent a total of about 20 hours in the chamber over the next few days during the treatment. I consider that his sports diving mentality, there is no pleasure without pain type of fun, must have contributed some way in getting the bends, even though he was within the tables.

The second case was a 30 year old Female with quite a bit of experience diving, again PADI trained, again after a weekend of very vigorous diving, who developed some rather vague symptoms which were attributed to slight decompression sickness. There was subsequently little or no improvement with recompression treatment. She was taking out a group of novices to teach them some of the aspects of tank diving. The first day was fairly easy with a lot of snorkelling. The dives were not actually deep enough to be included in the tables, so it was a fairly easy day's diving. But there was a lot of snorkelling, a lot of activity. The second day she worked very hard in and out of the water all the time. She was looking after lots of people jumping in and out of the water. She was quite cold as she was not wearing the right gear or enough of it. And she "bounced" twice, that is two quick trips to the surface and back down again. Looking at the US Navy tables one need not count the first day because the dives were more than 12 hours before those on day 2. Using the US Navy repetitive dive tables she was just on the no decompression limits with her stated depths of 40, 50 and 60 feet. If one used the Royal Navy tables which are more conservative, she had exceeded the no stops limits. But if, as one should do, one adds 10 feet, for each adverse factor such as cold exposure, doing a lot of work and bouncing twice, it turns out that she was well over the no stop limits. Even using the less conservative US tables she should have made stops of ll minutes at 20 feet and 48 minutes at 10 feet. In my opinion she had gone well over the no stop limits.

On day 4 she presented with very vague symptoms, mainly a pain in the back of the neck. She felt unwell slightly. Her examination was quite normal. This pain was difficult to elucidate. She said she had actually noticed it on the bottom during the last (night) dive. She was with a group of novices that night and she bent over to pick up a torch that had dropped and felt this pain come on. Suddenly the torch was floating up to the surface and so was one of her novice divers, shooting up with the torch. That led her to do her second 'bounce' It was decided that there was insufficient evidence for a clear diagnosis of decompression sickness in view of the vagueness of her symptoms and the absence of any signs at all. Although she had obviously gone over the no-stops limits we felt that the patient rather than tables should be treated. She was treated overnight with oxygen by face mask. She was discharged the next day with instructions to take it easy and return if she had any further problems or return of her symptoms. Return she did on day 9 with persistence of her symptoms, pain in the neck and generally feeling a bit unwell. It was considered then, after some discussion with Des Gorman of the School of Underwater Medicine, that she might well have been suffering from decompression sickness, slight as her symptoms were. So recompression treatment was organized. Although there was some suggestion of improvement here and there, generally speaking I have to say that she did not really improve very much. She still has some pain in the neck and still felt a little unwell at the end of her treatment.

Where does that leave us? The decision to recompress was based in some way on a theoretical understanding of what happens with neurological bends. It is thought that there

are micro-emboli of gas bubbles in capillary networks in the spinal cord and subsequent oedema and ischaemia of the spinal cord. Some people would clearly argue that this should be treated quite aggressively and vigorously with early recompression. Should decompression be used diagnostically in this regard? That is another possibility, another question to raise.

Another big factor that comes up is the cost benefit of recompression when one considers the enormous increase in the amount of sports diving that is being done and the big demand that a recompression treatment makes, particularly on human resources, not to mention the cost in dollars. In North Queensland where the human resource commodity is spread so thinly it is quite a demand. I think these cases, particularly the latter case, raises that important point.

These two cases of decompression sickness, one with obvious demonstrable neurological lesion where the US Navy, less conservative, tables were not exceeded but where the guy had obviously thrashed himself around that weekend and really overdone things, the second have a very marginal case where the diagnosis was in doubt and the treatment was not effective in that it did not improve her, ie. suspected bends only, where the US tables were obviously very well exceeded, leave us with the dilemma that we have always been in. Just what value are decompression tables in helping us decide when to treat someone and just how should we be interpreting them?

DECOMPRESSION SICKNESS AN OVERVIEW

Bart McKenzie

I have to try to compress decompression sickness into 25 minutes, which is going to be no mean feat.

To start with there are a few basic physical principles. The first one is the concept of pressure. There is about 100 km of atmosphere above us and that exerts a certain amount of pressure which is called one atmosphere pressure. Ten metres of sea water exerts the same pressure as one atmosphere. So at 10 metres there are two atmospheres of pressure acting on the diver. At 20 metres there will be a pressure of three atmospheres, and so on. A diver does not have to go very deep before there is a considerable pressure acting on him or her.

Now to mention a few gas laws. The first one is Boyle's Law which states that volume varies inversely with pressure. If one takes an inverted open jar and pushes it down under the water, as the water pressure increases the volume will decrease. This has a lot of importance in the treatment of various things in diving medicine. If a diver has gas bubbles in his tissues and one applies increased ambient pressure to the diver then the bubbles will decrease in size. This may eliminate his symptoms. If the diver goes up in an aeroplane while he had bubbles in his tissues, the bubbles get larger. This has relevance to the transportation of divers. If he was breathing from compressed gas equipment and took a breath at some depth and came to the surface holding his breath, then that would spoil his whole day. The next concept is Henry's Law. When gas is in contact with a liquid some of the gas will dissolve in the liquid. Double the partial pressure of the gas over the liquid then twice as much gas will be dissolved in the liquid at equilibration. So when a diver is breathing compressed air under water nitrogen is taken up by his tissues and that has certain consequences which I will go into later. An important concept is diffusion. If someone was to pass flatus on one side of the room then it would not be long before people on the other side of the room were looking accusingly at each other. That is the process of diffusion. You can use diffusion and diffusion gradients in the treatment of decompression sickness by modifying gas partial pressures. Another important law in diving is Murphy's Law which states that if something can go wrong it will go wrong, usually at the most inconvenient time.

I would like to stress that diving is great fun. However, like all things that are great fun it has some drawbacks. One of the big drawbacks of diving is decompression sickness. A sports diver breathing under water has a demand valve which delivers gas to him at a pressure which is roughly equivalent to the ambient water pressure. So the deeper he goes the higher partial pressure of nitrogen he is breathing. This nitrogen is taken up. It equilibrates instantly in his alveoli and then it is delivered by the blood to the various tissues. Tissue uptake of nitrogen is dependant on several factors, the main one being the blood flow of the tissues. So tissues that have a high blood flow take up nitrogen quickly and equilibrate quickly. While tissues that have a low blood flow do not equilibrate so quickly. When a diver comes back to the surface he has an excess of nitrogen dissolved in his tissues. If the partial pressure of nitrogen exceeds a certain critical limit the nitrogen will come out of solution and form bubbles. This is analogous to the situation of carbonated beverages. If one looks at a bottle of champagne before taking the cork out there are no bubbles in it. There is carbon dioxide in the bottle but it is dissolved under pressure and the cork holds the pressure in. When the cork is taken out the pressure is released, the bubbles come out of solution quickly and everybody has a good time. But divers with bubbles do not have a good time.

When diving with compressed air equipment started about 150 years ago the divers found that if they spent a long time under water, especially at great depth, they would develop the symptoms of decompression sickness. They also figured out by trial and error that if they came up in stages, rather than coming straight up, that could minimise the symptoms and sometimes stop them from getting decompression sickness altogether. So there were various ad hoc decompression routines built up over the years. However early in this century Haldane worked out some decent decompression tables which would allow divers to dive to practically any depth that they wanted to, at that time, and come back using decompression stops and not get decompression sickness. He based his tables on two basic hypotheses. The first was that the gas uptake by a tissue, or gas uptake by the body occurs in an exponential fashion and also that gas elimination occurs in an exponential fashion. He also dreamed up some hypothetical

tissues in the body and worked out his tables on the basis that there were probably half a dozen tissues in the body which took up nitrogen at different rates. Using exponential equations he could work out at any given time, for any given depth, what the partial pressure of nitrogen in these tissues would be. His second hypothesis was that divers could withstand a two to one difference in pressure between the pressure of nitrogen in the tissues and the ambient pressure without developing decompression sickness. The reason he came to this conclusion was that he observed that divers could spend almost an unlimited amount of time at depths less than 10 metres and come straight to the surface and they did not seem to get decompression sickness. The pressure at 10 metres is two atmospheres and the pressure at the surface is one atmosphere, so he concluded that divers could go from two atmospheres to one atmosphere and not get bent. He then extended that to suggest that maybe they could go from six to three or from four to two and so on. He worked out his tables on that basis.

During a dive the tension of nitrogen in the tissues gradually increases. In the fast tissue it increases quickly and in the slow tissue it increases slowly. When the diver starts to come up nitrogen starts to be eliminated from the tissues. But the diver will get to a stage where the ambient pressure is half the pressure of nitrogen in the tissue that contains the most nitrogen, which is the fast tissue. According to Haldane's hypothesis, if he is not to get decompression sickness, he has got to stop there. He spends a bit of time at that depth and waits for the partial pressure of nitrogen in the tissues to drop, so that he can come up a bit more and a bit more. So he comes up in stages waiting for the partial pressure of nitrogen in the tissues to drop so that it can be eliminated rather than forming bubbles. The critical tissue depends on time. Initially it is the five minute tissue. But further into the decompression routine, it becomes the ten minute tissue and if he stays long enough it might even become the seventy-five minute tissue. When he stops at the first stop, although the fast tissues are eliminating nitrogen, slow tissues such as the seventy-five minute tissue, are actually taking up nitrogen. Even when the diver is doing his decompression stops, some of his tissues are actually taking nitrogen up, not eliminating it. When they tried the Haldane tables out in practice they worked reasonably well, but they were not totally effective. The US Navy got hold of them and modified them a bit. In fact the US Navy tables that most divers use these days are based on the Haldane tables with some modifications that were made, basically by trial and error, by the US Navy.

The unfortunate thing about Haldane's tables is that both Haldane's hypotheses were totally invalid. It is quite amazing that the tables work at all. The first hypothesis that the uptake and elimination is exponential is invalid because we now know that when a diver comes back to the surface, if one puts an ultrasonic bubble detector on the diver's legs or on his chest, it will detect bubbles during routine dives, especially deep dives, even when the diver does not get decompression sickness. There is bubble formation in the tissues, but they do not get symptoms. So his hypothesis that the elimination of nitrogen from the tissues was exponential was quite invalid because as soon as you get bubbles in the tissues, that completely disrupts the dynamics of nitrogen elimination. The second one was this 2:1 ratio hypothesis. We now know that if divers spend long enough at 10 metres and then come to the surface they will get decompression sickness. There have been lots of cases described now of divers who have saturated at 10 metres and come to the surface and got bent. So the tissues cannot even stand a 2:1 reduction in pressure without producing decompression sickness. Although the tables work to some extent and stop people getting symptoms of decompression sickness they do no stop bubble formation in the tissues.

Various workers over the years have recognized that Haldane's concepts were fallacious and have tried to improve on them. They worked on various other theories for decompression and worked out tables. Oddly enough these other tables were not much more effective than the modified Haldane tables. The important thing that I want to stress after all this is that ALL THE TABLES ARE UNRELIABLE. It does not matter which tables one uses, the Royal Navy tables, the US Navy tables, or any other tables for that matter, they are not completely reliable. Divers find this idea heretical. They seem to have blind faith in these tables. They think that if they follow the tables they will be OK, and usually they will. We certainly urge them to follow the tables, but you cannot bet your life on them. Divers sometimes quote US Navy figures. The US Navy claim a less than one per cent failure with their tables which is pretty good. But if one looks at the kind of diving that US Navy divers are doing, one finds that they never push their tables to the limit. Most of the diving in fact is just odd jobs around ships. They clean the propellors on ships and they dive down to the bottom of harbours and pick up things that have been dropped off the ship. They tend not to do a lot of deep diving, they do very little repetitive diving, and they employ fudge factors. When they start to get to the fixed points of the tables, they start fudging. They add a little bit to the depth just to be on the safe side. And they also add a little bit to the time. Part of the reason for adding a bit to the time, I think, is the fact that they get paid a bonus for the time that they spend in the water. But it works both ways, it is a good arrangement, by putting the diver on to a longer decompression schedule it makes the tables safer and they get a bit more money.

Sports divers if they are sensible, should employ fudge factors too. They should not trust the tables. They should add a little bit onto their depth, a little bit onto their time and decompress accordingly. That is a good practice. But a lot of sports divers do not. A lot of them want to spend as much time in the water as they can and get the most out of their weekend. So they tend to dive deep. They tend to push the tables. They do not employ fudge factors. They use depth gauges to measure their depth. Incidentally how many sports divers calibrate their depth gauges? When they buy one off the counter, do they calibrate it then? If they have had it for a year or two do they ever check the calibration on their depth gauges? It is a real enlightenment to take a sample of divers' depth gauges and calibrate them. They can vary quite enormously. When sports divers are using inaccurate depth gauges they are pushing the tables to the limit. They do not know accurately how deep they have been, even if they watch their gauges. So one cannot

extrapolate the US Navy safety figures to sports diving.

There are other predisposing factors to decompression sickness apart from being a sports diver, which is a strong predisposing factor. They are things like age, older people tend to get decompression sickness more than younger people. Diving in cold water tends to be a predisposing factor. If the divers work hard during the dive, that tends to increase nitrogen uptake. Divers who are overweight, although it is good insulation, it is not good for preventing decompression sickness. Females have about a threefold increase in decompression sickness, compared with males. There are other predisposing factors. Diving at altitude, if one uses the ordinary tables in a mountain lake decompression sickness is much more likely. There have to be some modifications to the tables with altitude. Bounce diving or rapid changes in depth during the dive are predisposing factors. Flying after diving, especially after decompression dives is likely to cause decompression sickness. If one flies high enough in high performance aircraft one can get decompression sickness, simply from the reduction of pressure with altitude. The same thing can happen in altitude chambers.

One of the important concepts in the pathophysiology decompression sickness is the concept of gas nucleii. It appears that there are little bubble nuclei throughout our tissues. These are microscopic pockets of gas that are found in little hydrophobic niches in the tissues. With nitrogen coming into the tissues because the diver is breathing compressed gas, the extra nitrogen will diffuse into these gas nuclei and make them bigger. If they get big enough surface tension effects will cause a little bubble to enucleate from that and go off into the blood or into the tissues. One can observe these gas nuclei if you have a look at a glass of beer. You will notice that the bubbles do not come out in a haphazard fashion, they come out as streams of bubbles from little points on the glass. These are bubble nuclei, gas nuclei, and they exist in the tissues. Another important pathophysiological point is that once a bubble forms in the tissues it then acts as a foreign body and it brings into play the coagulation mechanisms. Platelets adhere to it and fibrin deposition occurs. Which can mean that even if the bubble is eliminated by recompression there is still all the gubbins that was around the bubble which can occlude blood vessels and cause space occupying lesions.

The symptoms of decompression sickness usually come on early. Fifty per cent of the symptoms will occur within the first hour and by 6 hours 90 per cent of them will have occurred. The earlier they occur the more serious the decompression sickness is likely to be. In the sort of divers that the people in the audience are likely to see, the most common symptom will be skeletal pain, the next most common will be cerebral symptoms and spinal symptoms as well. The pain is usually in the region of the joints, generally the shoulder joints. There are other joints which often produce symptoms. Often the symptoms occur in the adjacent joints, for instance the right shoulder and the right elbow, or the right elbow and the right wrist. The pain is of an aching quality and it gets worse with time and it hurts to move the joint. Especially after deeper dives, the divers are likely to present with neurological symptoms. The most common one is symptoms suggesting a bubble in the cerebral region and they can get just about any constellation of symptoms. Very commonly they get a headache, but they can get sensory disturbances over varying parts of the body or they can get motor disturbances. Sometimes they can get cerebellar symptoms with ataxia. They will often get spinal symptoms with paraplegia. In sports divers you will never see vestibular symptoms due to decompression sickness. This is something that is confined, as far as I know, to helium divers. So if you have got a diver with vestibular symptoms watch out, you may be missing something else, like barotrauma to the ears for instance.

Slow tissues tend to produce skeletal type bends. If a diver does shallow dives he tends to get slow tissue skeletal bends and if a diver does deep dives, deeper than about 80 feet or so, he is likely to get fast tissue bends which are neurological symptoms. If he does a short deep dive he will probably only get neurological symptoms. If he does a long deep dive he will get neurological and skeletal symptoms because he will put nitrogen into both his slow and his fast tissues. If he only does a shallow dive he is unlikely to get cerebral symptoms. One is unlikely to get cerebral symptoms from a 60 foot dive. The exception to this is the spinal cord, you seem to get spinal cord symptoms from just about any kind of dive. So I guess the spinal cord is probably a slow tissue, even though it is nerve tissue. One can work out the treatment of decompression sickness from first principles. Putting the diver in a recompression chamber and increasing the ambient pressure will make the bubbles smaller and that, hopefully, will relieve his symptoms. The sooner it is done the better because this avoids the secondary effects of coagulation, etc. The advantages of recompression, are to make the bubbles smaller and to increase the surface area to volume ratio which aids diffusion into and out of the bubble. High partial pressures of oxygen help increase the gradient encouraging nitrogen to leave the body.

I have run out of time. I would like to emphasise two points. The first is that in order to make a diagnosis of decompression sickness you first have to know that the patient has been diving, otherwise you can miss the diagnosis. The second point is that you can always get advice from the Navy. They have someone on call 24 hours a day, 7 days a week. If you have any diagnostic problems or if you want someone treated the Navy can organise that with the co-operation of the Airforce. The telephone contact number in working hours, Monday to Friday 0800-1600, is (02) 960 0333. Out of working hours it is (02) 960 0321.

SPUMS ANNUAL SCIENTIFIC MEETING 1985

This will be held on Bandos Island in the Maldives from Thursday 18 April to Wednesday 24 April 1985. Dr Carl Edmonds has accepted an invitation to be a guest speaker.

Further details on page 40.

IN-WATER OXYGEN RECOMPRESSION THERAPY FOR DECOMPRESSION SICKNESS

John Knight

The correct treatment for decompression sickness is recompression in a multi-man chamber. This chamber should have at least two compartments. A multi-man chamber is necessary so that an attendant may be in with the sufferer, and the two compartment chamber is necessary so that the doctor may come into the chamber, examine the patient, and exit.

Unfortunately, chambers like this are not too common in Australia. At the last count, the available multi-man chambers were:

one at Townsville, at the Australian Institute of Marine Science;
one at HMAS Penguin in Sydney;
one at Prince Henry Hospital, in Sydney;
one at Mallacoota, in north eastern Victoria, owned by the Fishermen's Co-operative;
one at Morwell, run by the National Safety Council of Australia, Victorian Division;
one at Braeside in Melbourne, run by the Melbourne Metropolitan Board of Works, but activated only when they are tunnelling in compressed air;
one at Hobart, run by the Royal Hobart Hospital;
one at Fremantle, at HMAS Leeuwin.

I believe there is a multi-man chamber available in Adelaide, but I am not sure of its location. Of course there are the multi-man chambers used by the diving industry both in Bass Strait and on the North West Shelf, but these are not normally available for the treatment of civilians. They are there for the support of the diving operations that are being conducted. If they are used for treating other people, those diving operations must stop. As this involves a considerable financial penalty, we will leave them out of the discussion.

Experience has shown that the success rate for the treatment of decompression sickness is much better if less than six hours has elapsed since the onset of symptoms. This is partly due to the blood-bubble interactions that occur and result in the bubbles in the blood acquiring a coating of protein material which converts them from being effectively "easily squashed ping pong balls" into "difficult to squash tennis balls". The ideal is to treat the diver as soon as possible after he develops symptoms.

If there is no chamber handy, one has to arrange transport to a chamber. As reduction in pressure will allow the bubbles in the patient's body to increase in size and make him worse, the patient should be transported as close to sea level as possible, preferably in an aircraft pressurised to ground level. That will take a while unless you are lucky and find one in the airport nearest you. It usually means the aircraft has to be flown from Australia to wherever the diver is and back again.

There are many places in Pacific where lots of people go diving which are more than six hours door-to-door to one of these chambers.

Some years ago, Carl Edmonds was worried about the number of people who were coming to HMAS PENGUIN having developed decompression sickness on a Pacific island and taking days to reach HMAS PENGUIN for treatment. He sat down and compared what was available to him and what was available on the island.

At HMAS PENGUIN there is a chamber into which the patient and his attendant can be put. The patient is given oxygen breathe and the normal practice is to take the patient to a depth equivalent to 60 feet of seawater (fsw). The patient then breathes oxygen for 20 minutes and air for five minutes, and so on until his symptoms are improved. About 85 to 90% of people improve and lose their symptoms on this minimum pressure oxygen treatment. Sixty feet of sea water is used as it is pretty close to the highest safe partial pressure of oxygen. The danger is oxygen-induced convulsions, acute oxygen poisoning. These convulsions, are provoked by exposure to oxygen partial pressures of two atmospheres and more. There is a pressure and time relationship. At 60 fsw, 100% oxygen has a partial pressure of 2.8 atmospheres and is safe to breathe, for most people, for periods of up to two hours. After a while the chamber pressure is reduced to the equivalent of 30 fsw and the process of oxygen and air breathing continued.

On a Pacific island there might be oxygen. There might be an airstrip that would take a Hercules or a Lear jet. There would certainly be water close by.

The problems of decompression sickness are bubbles which are usually treated by having their size reduced by recompression and oxygen breathing. There is always tissue anoxia which benefits from hyperbaric oxygen. All divers become dehydrated during their dive from the effects of immersion displacing blood from the legs and abdomen into the chest, so triggering stretch responses from the great vein volume receptors, although the blood volume is still normal. Decompression sickness causes leaky capillaries, among other problems, so the diver becomes further haemoconcentrated and dehydrated. He needs fluids. Two aspirin will discourage platelet aggression on the bubbles. The most important part of the treatment is recompression.

Recompression breathing air adds nitrogen to a body already overloaded with nitrogen. Therapeutic recompression in water breathing air is condemned by all diving medical authorities as it takes many hours to perform properly and exposes the diver to the risks of hypothermia. Most attempts by divers end disastrously.

There are advantages in breathing oxygen. The patient is no longer taking up nitrogen and he has the highest possible gradient for excreting the nitrogen load. This gradient increases considerably if the patient is in the water breathing oxygen.

PRINCIPLES

So Carl Edmonds decided that the divers who took days to reach his chamber would be better served if they were recompressed in the water while breathing oxygen soon after the onset of symptoms. He chose a depth of 9m (30

feet) because oxygen convulsions are unknown at this depth in people at rest. There have been convulsions in divers working hard in oxygen rebreathing sets at this depth, but investigations have always shown a raised PCO_2 in the set. The convulsion threshold of oxygen is lowered by a raised PCO_2. The diver having in-water oxygen recompression is still and has a normal PCO_2.

The advantages of in-water oxygen recompression at 9m can be summarized:

1. No nitrogen is added to the tissues during treatment.
2. There is a large gradient for nitrogen excretion.
3. The bubble volume is almost halved.
4. The diameter of circular bubbles is reduced by about 20%.
5. There is increased tissue oxygenation.
6. There is no risk of oxygen toxicity.
7. There is no risk of decompression sickness for the attendant.
8. The wetsuit is still effective insulation.
9. It can be instituted relatively quickly anywhere there is 9m of water.

There are disadvantages. I must emphasise that it is not ideal treatment. It is a treatment that is better than waiting around for six hours for an aircraft to fly you to a treatment centre that is properly equipped.

The water must be warm. It is not a treatment to be indulged in in cold water. Some three or four years ago this treatment was tried at Heron Island. After half an hour the patient complained bitterly of the cold, and insisted on terminating his exposure to pressure. By then he was improved but had not completed the treatment. It is not a good idea to add hypothermia to the problems of decompression sickness.

There are problems of breathing high partial pressures of oxygen. Vasoconstriction is induced. The immersion of a human in thermoneutral water increases his peripheral circulation, but thermoneutral water is not all that common in the ocean. It is more likely that the vasoconstriction induced by cold from the first dive and the heat loss from the treatment in water will induce vasoconstriction, so slowing the elimination of nitrogen from the tissues.

However, when compared with the problems of late treatment in-water oxygen recompression has its place where the water is warm and when transport to the nearest recompression centre will take more than six hours.

In-water oxygen recompression has been used on quite a number of occasions, with success, including at least one person who was unconscious when lowered into the water with his buddy alongside. He woke up under pressure and made a complete recovery from his central nervous system decompression sickness. So far I have not heard of any failures, and that news would go round the diving medical world very fast indeed, as many people consider that in-water oxygen recompression therapy is misguided, to put it mildly.

Certainly, if I was to develop decompression sickness and it would take more than six hours to get me to a recompression chamber, I would opt, if I was in the tropics, for the in-water recompression therapy immediately, on the basis that rapid treatment is much more effective than delayed treatment.

The requirements for in-water oxygen recompression are simple.

1. A large (F or G) size oxygen cylinder.
2. An oxygen reducing valve (regulator) set to deliver at least 80 psi. The regulator from an oxy-acetylene outfit will do very well as it delivers a higher pressure than the usual medical oxygen regulator, which is set to 60 psi.
3. 12 m of high pressure hose to connect the regulator to.
4. A full face mask.
5. The patient wears a full wet suit, including a hood.
6. A rope marked in metres or feet so that the patient's depth is known.
7. An attendant in the water with the patient.
8. A support for the patient.
9. A communication system both between the patient and his attendant, and between the attendant and the surface. The patient can speak quite comfortably if he is wearing a full face mask because there is an airspace for him to speak into and it is possible, if the buddy holds his breath for a moment, to hear quite clearly what the diver is saying. The other way around is more difficult, and an underwater slate is a good idea. The simplest method of communication between the attendant and the surface is to have another standby attendant who can go down when signalled for and relieve the existing buddy so that he can go up and give a verbal report.

A full face mask, in my opinion, is essential, although I know this treatment has been carried out without such things, because the full face mask allows the person to speak, it is less tiring than holding a regulator in the mouth for three hours and should the patient go unconscious and go on breathing, he will not drown. When using a normal face mask and a separate regulator, if somebody goes unconscious, their jaw muscles slacken and the regulator falls out of their mouth. They may or may not go on breathing. If they go on breathing, they are likely to drown.

The procedure is to ready a seat for the diver to sit on. This is more comfortable than sitting in the bight of a rope.

The diver must wear a wetsuit, including hood, because he is probably already cold from his dive, and is certainly going to get cold sitting still even in tropical water, because he will not be generating any noticeable amount of heat. Shivering is undesirable as it increases his oxygen usage, makes his muscles move and will precipitate the formation of extra bubbles in his body. The diver has to be overweighted so that he will stay at the depth chosen. If he is sitting it is a good idea to have weights on his ankles so that they do not tend to float up. This means that the diver must be attached to a safety line which is attached to the boat or jetty so that he cannot sink further than the length of the line. So we now have two lines. One to hold his seat, and one to catch him if he slips off.

Then there is the full face mask and the oxygen cylinder on the boat or jetty.

There has to be a team to carry out the procedure. At least one person to watch the time, the depth and the oxygen supply. Two attendants for the patient. More people are an asset.

The patient and his attendant disappear over the side and the patient is lowered to a depth of 9 m.

He then stays there for 30 minutes, regardless of how soon his symptoms are cured. In most cases these symptoms have been cured within the first 30 minutes. If the symptoms are not completely cured after 30 minutes the patient spends another 30 minutes at 9 m. If he had a neurological bend he spends at least 60 minutes before the ascent is commenced. If there are still symptoms remaining at the end of the hour, the time is extended by another 30 minutes.

So after 30, 60 or 90 minutes the return to the surface starts. Ascent is at the rate of 1 m every 12 minutes, or alternatively, for those who date from the pre-metric days, one foot every four minutes. You will have noticed that one foot every four minutes is slightly slower than 1 m every 12 minutes, and I think that the slower rate of ascent is to be recommended. Also the ascent of a foot at a time is nearer a lineal decompression than an ascent of a metre. For a given time span lineal decompression is a better method than a staircase decompression. At this rate the ascent takes about two hours.

If symptoms recur during ascent the ascent is halted for 30 minutes. I have never heard of this being necessary.

If the oxygen runs out the patient is brought straight to the surface. NEVER give the patient compressed air.

When the diver is out of the water, he is then given oxygen to breathe. Alternating oxygen on for an hour, oxygen off for an hour for twelve hours.

I have used the Edmonds table in a single man chamber. It seemed better at the time to compress the patient on oxygen in a small single man chamber than to lower him into the sea as dusk approached as we would have had to go some way out from the island of Moen to get the 9 m of water we needed. It was a wrong decision for the bus ride over a very pot-holed road converted him from being a person with mild neurological decompression sickness, stocking anaesthesia of both feet, into a paraplegic with the left arm also paralysed, who was having difficulty with his breathing. However, within half an hour at thirty feet with oxygen, he was able to move all three paralysed limbs. He was then taken to 60 feet but had little further improvement. Nitrogen loads in the body take a long time to decay. This man had last dived more than 48 hours before he appeared, mildly drunk, at the hotel that we were staying in. Travelling for 15 minutes over a very pot-holed road converted him from a mild case to a very severe case of decompression sickness so that there must have been a lot of gas available to form enlarged bubbles, even forty eight hours after diving.

Any procedure that gets this extra gas out of the body is to be recommended, and if somebody develops decompression sickness they should be given first aid consisting of oxygen to breathe and fluid to drink, because all divers are slightly dehydrated by the end of a dive, and two aspirin to inhibit platelet aggregation. As soon as this has been done, the buddy races to the telephone and gets advice as to what should be done next. Which in most cases on the Australian mainland, is to transport them immediately, at low level, to the nearest chamber.

However, if you are on the island of Truk in the middle of Micronesia, there is no quick arrival at the nearest chamber in Guam. It would be a very expensive trip indeed to charter a Lear jet, if there is one at Guam, to fly over to Truk and then pick up the patient and fly him back to the US Navy Hospital at Guam. Far better to arrange for an in-water recompression, in warm water with good visibility and watch the symptoms disappear.

TRANSPORTATION OF PATIENTS SUFFERING FROM DECOMPRESSION SICKNESS

Chris Acott

I am keenly interested in the transportation and retrieval of patients. I will discuss general aspects of patient transport with special reference to the transportation of patients with decompression sickness (DCS).

One would always like to know the overall general medical condition of the patient, however this is often hard to obtain. Is the patient deteriorating? Will he need specialized help before transport? Will transport at this stage adversely effect the patient?

DCS patients are usually stable, and need to be transported to the nearest recompression chamber as soon as possible. However the patient may be unconscious, fitting, or in respiratory difficulties, or have a spinal cord lesion that is progressing rapidly.

TABLE II

TRANSPORT MODES AVAILABLE

ROAD TRANSPORT

ADVANTAGES	DISADVANTAGES
Patient only transferred in and out of ambulance	Slow
Able to stop en route to perform procedures	
Ample room and lighting	
Adequate supply of everything	
Cost effective	
Able to transport in all conditions	Subject to road conditions
Altitude usually not a problem	But the ambulance may have to travel over a mountain range which is greater than 1000 ft. No way of knowing the altitude that you are at.

AIR TRANSPORT

FIXED WINGED AIRCRAFT

ADVANTAGES	DISADVANTAGES
Shorter transit time	Need airstrip May take long time to mobilise aircraft Subject to weather conditions
No peak hour traffic problems	Patient may be transferred several times, ie. ambulance to plane and back etc.
Adequate oxygen supply 2 x D cylinders, 1500 L each giving oxygen at 15 L/min for 3 hours.	
Can fly at sea level if altitude a problem	May be hazardous Expensive in fuel
Can use aircraft pressurized to ground level	Requires time to mobilize such an aircraft
Adequate space and lighting. Can use ECG	Inability to change therapy enroute Noise levels make monitoring of breath sounds difficult. BP only by palpation.

HELICOPTERS

ADVANTAGES	DISADVANTAGES
No airstrip needed Practically land anywhere	Need down draught to take off
More stable than fixed winged aircraft in adverse conditions	
Cruising speed 110-130 mph	
Adequate room and oxygen supply	The average Bell helicopter not as roomy as the road transport
Flights can be kept below 1000 ft	
Bad weather only limits flights 10 days per year, in the Sydney area	
Vibration and noise not a problem	Vibration may perhaps cause extra bubbling in DCS but there is no evidence of this.
Not subjected to increase in vibration level at take off and landing	Expensive

In the majority of cases First Aid can be administered by the diver's buddies or other non-medical people. The recommended routine is to give 2 aspirin tabs, give 100% oxygen, give 2 litres of fluid orally or intravenously (IV) and keep the patient still.

Spinal cord lesions will need a catheter before transport, and an IV dose of dexamethazone to help in the treatment of spinal cord oedema.

Mobilization of resources is essential. Notification of the nearest recompression chamber, and discussion with them about the general condition of the patient is necessary. The total time for transport is very important. This time including the time needed to mobilize the ambulance. Other factors are the distance to be travelled, the weather and road conditions. Fog, winds and rain will lengthen the transport time, time needed to get to the patient. When air transport is used the time needed to travel to and from airstrips has to be included.

Other things that must be considered are the ability to change therapy en-route, the ability to monitor the patient en-route, an adequate oxygen supply, proper suction facilities and the capabilities of the escort. One must consider whether the effects of transport will adversely effect the patient, ie. vibration or altitude.

TABLE I

GENERAL CONSIDERATIONS FOR CHOOSING TRANSPORT MODE

General condition of the patient

Will transport at that stage adversely effect the patient?

Ability to change therapy en route

Ability to monitor the patient

Adequate supply of drugs and oxygen for the journey

Distance to be travelled

Communications

Actual time of transport
(a) time to mobilize transport
(b) time to travel the distance

Altitude is important when transporting DCS patients. The bubbles in the body will obey Boyle's Law, Pa1/V, therefore any reduction in pressure, as happens with an increase with altitude, will cause an increase in the size of the bubble, and hence make the condition of the patient worse. At 8000 ft (commercial aircraft) the bubble volume will increase 25-30%. With increasing altitude the partial pressure of oxygen will decrease. At 8000 ft the atmospheric pressure will be 567mm Hg, the alveolar O_2 66mm Hg. All patients will need O_2 by mask at the highest concentrations possible. Using a flow of 15 litres a minute (lpm) a D cylinder will last 90 minutes.

Several modes of transport are available, road, aeroplane or helicopter. Table II lists the various advantages and disadvantages of each method.

The definitive treatment of all cases of DCS is recompression. The sooner that it is done the better. Optimally the best form of transport would be under pressure, so that the treatment has already begun during transit. A two-man portable recompression chamber is the safest. Here an attendant is present with the patient, so he can administer therapy en-route. These chambers can also lock onto the mating larger chamber, so allowing transfer under pressure. One man chambers are available, but once the patient is in them, it is impossible to get to the patient unless the chamber is decompressed. They should not be used for transporting patients. Failing a portable chamber, the patient is best served by keeping him at sea level (1ATA), giving him 100% oxygen and transporting him rapidly to the nearest chamber.

Aircraft pressurized to ground level can be hard to obtain. An RAAF Hercules would take considerable time to mobilize to Rockhampton. Commercial aircraft are only pressurized to 8000 ft. There are small fixed winged pressurized aircraft but they are not readily available. We are very fortunate, indeed, here in central Queensland that the Ambulance Service has a pressurized-to-ground-level small aircraft for patient transport,

There is one other problem. Often DCS patients are in a lot of pain. The people attending him may be tempted to give him ETONOX (Nitrous oxide and oxygen mixture) to breathe. Please DO NOT. ETONOX will only make his condition worse as the nitrous oxide diffuses into the bubbles and makes them larger.

Transportation of these patients should be done quickly, safely and with consideration of the problems involved. Mobilization of resources is important as is using them to the best possible advantage to the patient, especially considering the major problem of DYSBARISM.

DISCUSSION

Dr J Williamson

The poor old sports divers really got it in the neck today. I would like to assure people, if they are not already aware of this, that these errors in diving technique and complications are not confined to sports divers. People who dive as part of their work are usually under close and strict supervision and that is very good. But left to their own devices they can get into heaps of trouble and they sometimes do.

There is no such thing as a safe dive so any doctor presented with a diver with symptoms, should not be put off by the fact that their apparent profile is well within the so-called limits of a diving table. There is no such thing as a safe dive. One must always remember that all divers are unreliable witnesses. This does not mean to say they intentionally set out to deceive but very often they quite often say and believe things which on closer enquiry are found not to be true.

I would like to challenge John Knight's statement that there is no danger of CNS toxicity oxygen treatment at 9 metres. That is quite untrue.

Dr J Knight

I did say that CNS oxygen toxicity had been seen at that

depth in closed circuit users. I am not aware of it ever having occurred at 9m in somebody who was not using closed circuit equipment in which the CO_2 level has risen. There may be a possibility of it but I have not heard of it.

Dr J Williamson

Well you have heard of it now because we had it in Townsville. I think the important point to make in this regard is that when you are looking at thresholds for anything, in particular oxygen toxicity, but also a lot of other things, a lot of the experience is based on divers sitting comfortably and dry and not working in chambers. But when you put cold, frightened, sick divers back into the water the thresholds are quite different.

Dr J Knight

Could I ask if your case appeared in the SPUMS Journal? If it did, some other explanations have been advanced. Carl Edmonds feels that it was a case of salt water aspiration not oxygen toxicity. But you saw it so you ought to know.

Dr J Williamson

There is no question of it having occurred. The sequence of events was just right down the line for oxygen toxicity. There was no question of salt water aspiration. Are you postulating that salt water aspiration can produce convulsions?

Dr J Knight

No. I would suggest that you have just given the explanation. Divers are poor witnesses. Unless you were actually in the water with him, if he shivered and shook it might well be interpreted as a convulsion by somebody who did not know better. I would not accept a convulsion unless he goes unconscious and he is seen to be unconscious by somebody who knows what unconsciousness is. I do not think most divers are in that category.

Dr J Williamson

As nearly as we can verify all that, all those conditions were satisfied. The people were quite expert in the diving field. As it happened, Vic Callan and I appeared on the scene within minutes after he was brought back to the surface. I think there is very little doubt that this man convulsed.

Dr I Gibbs

I would just like to say a little bit in support of what Dr Williamson just said. I'm ex-Royal Navy and I spent a couple of years at HMS VERNON which is the main Royal Navy diving school in Portsmouth. We used to see a lot of oxygen toxicity in Navy divers trained for the highest levels of professional skill. They were all tested in the chamber so their oxygen tolerances were known. We knew that they were never diving more than 30 feet because there was not more than that to dive in. They may have been exerting themselves and they were probably cold and so on but I would suggest that the incidence of oxygen toxicity amongst the general population would probably be considerably higher at shallower depths than applied to these Navy divers.

Dr J Knight

Are you saying that the clearance divers developed CNS oxygen toxicity without getting raised PCO2 while they were swimming about.

Dr I Gibbs

No. What I am trying to say is that there was a significant incidence of oxygen toxicity at less than 10 metres in blokes who had been specifically tested to prove that they had not got a low threshold to oxygen. I would suggest that the normal civilian population would be more prone to oxygen toxicity than the naval divers. I have reservations myself about the desirability of putting, as Dr J Williamson says, tired frightened people in the water at 9 metres and hoping that they will not have a fit.

Dr J Knight

That is one reason for having the full face mask so that they will not drown if they go unconscious. After all the Royal Navy users full face masks so that their divers can go unconscious quite safely. In water oxygen recompression is not ideal treatment if there is a chamber within easy reach. It is for those people who are not close to a chamber. The great majority of people who get bent do not have neurological bends and most of them cure quite quickly.

All I can say is if there had been any major problem, it would have been round the diving medical world very fast, because few people approve of this treatment. People have spent a lot of time unsuccessfully trying to find cases where it did not work, or had to be abandoned, and the patient was worse as a result.

I think it should not be used off the Queensland coast where one can get a helicopter out and be taken to Townsville and recompressed. On the other hand in Madang the time to get a Lear jet that can pressurise to surface over to Madang and then back to Townsville is going to be more than 6 hours in all probability.

In sheltered, warm water I would prefer to be recompressed on oxygen in the water rather than wait more than 6 hours for treatment. I am not telling everybody to do it. I am saying that I think it is safe enough to risk my life being treated in that way when the alternative is a long wait for treatment.

Question

If you used a Magill circuit, even though it is slightly less convenient than a Mapleson C circuit, you would probably be able to cut flow down to 5 or 6 litres a minute (lpm) rather than 15 lpm, which would give you three times as much oxygen for each cylinder.

Dr C Acott

It is difficult to put Magill circuit into an oxygen cylinder in an aircraft. I take a Mapleson C with me and an ordinary Hudson mask. I have high flows on it, around 13 to 15 lpm. I was using 15 lpm as an example. You have got to know how far you have got to go and how much oxygen you have got on board. It is no use getting up there and finding out you have run out of oxygen.

THE DEVELOPMENT OF THE NATIONAL ASSOCIATION OF DIVER MEDICAL TECHNICIANS (NADMT) IN THE US

Jim Joiner

In the early 1970s, the critical worldwide energy shortage motivated the oil companies to explore and drill for petroleum products in the waters offshore in many countries. As the need for petroleum gradually increased, the drilling activities and consequently the diving activities took place in ever deepening waters. Often the drilling rigs were located in isolated sections of the world far from adequate medical facilities or hyperbaric specialists.

The need to improve the level of on-site medical management for diving accidents and illnesses increased with the rapid move into these hostile working environments. By 1973-74, the diving contractors and the diver training institutions, as well as the unions and government agencies, were becoming alarmed by the rising number of accidents. They were aware that in many cases the accidents were aggravated by lack of proper treatment, misdiagnosis and the inability to provide immediate medical care. While it was recognized that the isolation of divers under pressure and the geographic remoteness of diving sites create unique difficulties that necessitate a high degree of medical and technical competence, at that time there were no criteria as to how these emergencies should be handled or by whom.

Too often the responsibility of accident management became part of the job of the diving supervisor who usually was poorly prepared to meet the demands of acute medical diagnosis, treatment or intervention. Some offshore rigs employed rig-medics, but most of these medics had had no special training in hyperbaric problems and treatment. At the time, there were few hyperbaric physicians and those who were qualified to treat diving accidents often were too far away to help in an emergency.

Therefore, at the urgent request of the diving contractors, divers and its parent company, Oceaneering International, the Commercial Diving Centre (CDC) (Now the College of Oceaneering) began to develop a new programme to train a selected group of divers and supervisors in diving accident management. The request for a training programme was fully supported by the Association of Diving Contractors (ADC).

Under the direction of Jim Joiner, Executive Director of CDC, a committee of educators and medical personnel met to develop a course outline, curriculum and training procedures that could prepare experienced divers and supervisors from the field to handle diving accidents and emergencies. The planning committee felt that the divers and supervisors would be completely supported by their respective employers in as much as the program had been requested by them. The planning committee consisted of Joiner and Dr Richard Scott, USC Medical Centre, Drs Paul Linaweaver and Hugh Greer of the Santa Barbara Medical Foundation Clinic and Dr David Youngblood, Medical Director of Oceaneering International.

The programme was designed to train the divers and supervisors to accurately and adequately diagnose diving and other related accidents, injuries and diseases and provide effective treatment (under the direction of a diving medical physician whenever possible) for up to 72 hours. The initial course was designed for participants who had had a minimum of 100 days of offshore work and preference was to be given to those divers who had past experience as a military medic or a civilian paramedic.

The pilot programme in 1975 was attended by 12 divers and supervisors who had the complete support of their employers, all active members of ADC. This pilot programme was carefully monitored and comprehensively critiqued by all the parties involved and declared a success. It was anticipated at this time that the new Diver Medic Training Programme would meet the needs of a separate section of the National Registry of Emergency Technicians and that certification of the diver medics would be merely a matter of formality.

Unfortunately, this was not to be the case. During the next six years the setbacks and obstacles encountered, accrediting the programme and certifying the diver medics were almost beyond belief. The first major barrier to formal accreditation was caused by the lack of clear legal interpretation of the diving medical aspect. Because no one was certain as to who had the authority to certify divers to work both domestically and internationally, no one would assume the responsibility.

Therefore, during the next six years, the National Registry certified diver medic graduates as Emergency Technician I, but refused official recognition of the diving medical aspect. This was equivalent to no recognition in the legal viewpoint of oil companies and diving contractors.

Although the programme was formally recognized by the Undersea Medical Society (UMS) in 1976, they decided not to accredit the programme or certify the divers as did the Occupational Health and Safety Administration (OSHA), the Bureau of Emergency Medical Services, the Health and Human Resource Administration, the Department of Transportation (DOT) and the United States Coast Guard.

In spite of these setbacks CDC continued to offer the diver medic training programme on an “as needed” basis. But as the formal recognition of the programme continued in abeyance and the US governmental agencies declined to mandate diver medics on offshore installations, the interest of employers in sending supervisors and divers to the programme declined.

By 1978, 124 divers had graduated from diver medic courses, of these 42 were company sponsored, 4 were union sponsored and 78 were individuals who elected to pay for their personal training. Economic circumstances then dictated that the classes be opened to new diver trainees. Therefore, the programme was modified to accommodate the changes in the experience and expertise of the programme participants. The programme was lengthened, basic training made more intense and a home study portion was added.

During the next two years other diver training institutions used the programme at CDC as a model to develop their own diver medic programmes.

Initially, programmes were begun at Ocean Corporation and Florida Institute of Technology. At this time, a Medical Advisory Group was formed to advise and oversee the training of diver medic personnel. The Medical Advisory Group was concerned that all diver medic training programmes should meet the minimum standards established by the group and use qualified instructors, have proper medical facilities for on-site training and that the diver medic trainees be properly recognized and compensated.

In 1979 the Undersea Medical Society agreed to provide formal accreditation for the diver medics provided the regulatory agency, the Department of Transportation, would provide curriculum approval. Before this could be accomplished, diver safety and health programmes involved with offshore diving operations was transferred from the DOT to the jurisdiction of the US Coast Guard. Repeated inquiries to the Coast Guard for official recognition of the diver medic curriculum resulted in a May 1980 decision that "... at this time, the US Coast Guard has no specific plans to introduce any regulation concerning Diver Medics, their training or certification or their required presence."

In 1981 the National Association of Diver Medical Technicians (NADMT) was formed for the express purpose of accrediting diver training institutions, and as the official certifying agency for diver medics, instructors and diver medic programmes. Within two years NADMT was formally approved by UMS and the Association of Diving Contractors.

The Board of Directors is composed of leading diver training educators, hyperbaric physicians and representatives of UMS and ADC. This group meets on a regular basis to plan for the improvement of the organization and to meet additional needs of the diver medic field. Currently the organization with the assistance of UMS is in the process of developing guidelines for re-certification and re-training of diver medics. Future plans call for the development of books, supplies and teaching aids for the training programmes. The outline and criteria for these materials have already been developed and will commence as soon as funds are available. This year NADMT published a Field Reference Guide Handbook for Diver Medics written by Dr Gordon Daugherty.

At the present time, Canada, England and a few other countries are in the process of developing their own national diver medic programs and respective associations for the certification and accreditation of divers and diver medic programmes. It is anticipated that within the next few years it may be possible to form an international qualification for diver medics. In this way, diver medics with recognized certification will be approved to work in territorial waters anywhere in the world.

The 1984-85 President of NADMT will be Jim Joiner, President of the College of Oceaneering. Anyone interested in learning more about NADMT or the training programmes offered for diver medics is invited to write to Mr Joiner at

The College of Oceaneering
Los Angeles Harbor
272 South Fries Avenue
Wilmington
California 91744
USA

DAN STARTS MEMBERSHIP DRIVE

DAN, operating with the new title Divers Alert Network, has begun a membership drive for individuals and corporations interested in diving safety.

Formerly known as Diving Accident Network, DAN is a national system of recompression chambers accessible through one phone number available nationwide, 24 hours a day. In an emergency, calling 919-684-8111 and asking for DAN connects a diver or his physician to a specialist in diving medicine who can advise correct early treatment and assist in referral to an appropriate treatment facility. Additional non-emergency information can be obtained by calling 919-6842948, Monday through Friday, 9 am to 5 pm EST.

Individual members of DAN will receive the "Underwater Diving Accident Manual," tank decals with the DAN diver's flag logo and emergency number, a newsletter of diving medicine in layman's language, and a membership card allowing dollar saving privileges. Membership fee is $10. Diving related businesses are asked to become Corporate members and will receive a number of free memberships and other benefits for their commitment to diving safety. For more information, please contact: The Divers Alert Network, Box 3823, Duke University Medical Center, Durham, North Carolina 27710.

Reprinted, by kind permission of the Editor, from TRIAGE, the newsletter of the National Association of Diver Medical Technicians, 5709 Glenmont, Houston, Texas 77081, USA.

LETTERS TO THE EDITOR

Sir,

I read with interest the SPUMS J 1983 (3), which brought to mind an editorial I wrote in 1973 regarding the abuse of the certification card that we have suffered in the US and Canada. In the past several years we have had lawsuits directed against the person who rented the cylinder for an uncertified friend. In two cases they were settled out of court, and another case went to court and a substantial judgement was given against the person that rented the cylinders. It takes editorials to bring such matters to public attention. I append a copy of what I wrote in 1973.

SHAME ON YOU!

An uncertified diver died today in 6 feet of water off the coast of San Diego. He made the "tele" and he made all the newspapers. He hurt his sport and he hurt the industry I make my living in, and he hurt all of my close personal friends that work for NASDS and all the advertisers that support this magazine. It upsets me. And it was someone's

fault that should have known better. The fellow that died wasn't a diver. He couldn't have been because he had never had a lesson in his life.

He didn't rent the gear from a store/school because he wasn't certified and he knew that the store wouldn't rent the gear to him unless he was.

But his buddy was certified. And his buddy rented the gear for him. That's what you call a Good Friend. He had a working regulator with a submersible (contents gauge) and a BC with a push button on it. All worked. The trouble was that because he didn't know how to use it, none of the tools that should have saved his life did him a damn bit of good because he did not have instruction on any of the super gear his buddy rented. 6 feet of water. If he had been 6 feet tall he could have stood up and walked to safety.

If you know anyone that is going to rent or get equipment for someone that is not certified, do him a favor. Call the doctor and get him committed to a hospital for the criminally insane.

John Gaffney
Executive Director
NASDS (USA)

These robust sentiments are as valid today as they were when penned in 1973.

Editor. SPUMS J

PROJECT STICKYBEAK

This project is an on-going 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

SPUMS ANNUAL SCIENTIFIC MEETING 1985

This will be held on Bandos Island in the Maldives from Thursday 18 April to Wednesday 24 April. The two guest speakers are Dr Straun Sutherland of the Commonwealth Serum Laboratories and Dr Carl Edmonds, founding President of SPUMS.

Flights to the Maldives leave on different days from the various capital cities so some members will have to arrive early at Bandos Island and some leave after the main party.

For those leaving from Adelaide and Brisbane the departure date is Saturday April 13th. There will be a 2 night stopover in Singapore, 10 nights in the Maldives and a one night stopover in Singapore on the way back to Australia, landing on Saturday April 27th.

From Perth the departure date is Tuesday April 16th. There will be a one night stopover in Singapore. The stay on Bandos can be 8 nights with a 4 night stopover in Singapore on the return journey or it can be 12 nights in the Maldives with a direct return to Australia. In either case Perth Airport is reached early on Tuesday April 30th.

From Melbourne and Sydney there is a choice of departure dates.

1. Monday April 15th, returning on

   (a) Friday April 26th which allows

      i) 10 nights in the Maldives, or

      ii) 8 nights in the Maldives with a 2 night stopover in Singapore either going or returning.

   (b) Sunday April 28th which allows

      i) 10 nights on Bandos Island with a 2 night stopover in Singapore either going or returning

      ii) 8 nights in the Maldives with a 2 night stopover in Singapore on both journeys

      iii) 12 nights on Bandos Island.

2. Wednesday April 17th returning on

   (a) Friday April 26th which allows 8 nights in the Maldives.

   (b) Sunday April 28th which allows

      i) 8 nights on Bandos Island with a 2 night stopover in Singapore on the way back

      ii) 10 nights in the Maldives.

   (c) Tuesday April 30th which allows

      i) 8 nights in the Maldives with a 4 night stopover in Singapore on the way back

      ii) 10 nights in the Maldives with a 2 night stopover in Singapore on the way back

      iii) 12 nights in the Maldives.

On the return journey it will be possible to substitute a 2 night stopover in Colombo for a 2 night stopover in Singapore.

The registration brochure should have reached you before this issue of the Journal. For further travel information, and copies of the brochure contact:

Allways Travel Service,
168 High Street,
ASHBURTON, VICTORIA, 3147.
Telephone: (03) 25-8818