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South Pacific Underwater Medicine Society Incorporated

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ARTICLES OF INTEREST FROM OTHER JOURNALS, GLEANINGS FROM MEDICAL JOURNALS AND COURSES ARE DETAILED ON THE BACK COVER

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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.

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

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

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

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

References

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

1 Anderson T. RAN medical officers' training in underwater medicine. *SPUMS J* 1985; 15 (2): 19-22
2 Lippmann J and Bugg S. *The diving emergency handbook*. Melbourne: J.L.Publications, 1985: 17-23

Computer compatibility

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

Editing

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

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

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Abstracts

Abstracts from the Undersea and Hyperbaric Medical Society Annual Scientific Meeting 1989

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PROJECT STICKYBEAK

This project is an ongoing investigation seeking to document all types and severities of diving- related accidents. Information, all of which is treated as being **CONFIDENTIAL** in regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organisation to increase diving safety through better awareness of critical factors.

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EDITORIAL

Over the last few months the Committee has been made aware that many people have erroneous ideas of the position of SPUMS on various matters. The problem seems to be that many in the diving community assume that, because a diving doctor is well known to them, his or her views are those of SPUMS. This is not so. SPUMS has two official spokesmen, the President and the Secretary. On occasion the Committee may authorise a member to represent SPUMS on an outside committee and speak for the Society on the topic covered by the committee's deliberations. On other occasions a member may be authorised to speak for the Committee on a subject for which the member has special knowledge. Just because a paper or opinion appears in the SPUMS Journal it does not mean that it is SPUMS policy. In fact those who assume this have failed to read the cover of the Journal where the following disclaimer appears. "All opinions expressed are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policy of SPUMS". In other words SPUMS views are ONLY put by the President and the Secretary, speaking in their official capacity.

Readers may have been puzzled to see the title "Queensland scuba divers and their tables" again on the cover. We apologise to Jeffery Wilks and Vincent O'Hagan for an editorial error which saw their table fail to make the page. It appears in this issue instead. The results portrayed are an indictment of current, and past, teaching of the use of decompression tables. Less than half of these certified divers could use their tables. The proportion should be 100% if they are being properly trained.

John Lippmann has two contributions to this issue. The first is an educational exercise, a critique of the problems that lead to four deaths of divers in Victoria. His comments will be of interest to every diver. The second is to be found on page 128. He has, once again, organised a raffle to raise money for the Diver Emergency Service (DES). His last raffle (two years ago) raised enough to purchase a mobile phone and this time he has rounded up some magnificent prizes including a week on board Mike Ball's "Spoilsport". DES is always in need of funds as government funding does not cover costs and, inspite of promises, not all the diving instructor organisations have been contributing regularly. Here is a painless way for members to contribute to DES and the chances are much better than the usual lottery as there are only 3,000 tickets at $ 5.00 each. So buy your tickets and support DES.

For those who are interested in asthma and diving there is summary of current views, in the form of edited extracts from the still-to-be-published 3rd edition of "Diving and Subaquatic Medicine". In the Letters to the Editor is a plea for help with an other chronic disease, insulin dependant diabetes. Although it is a diving medical non-starter there are diabetics who dive. No one knows their experiences. We hope all those diabetics on insulin, who have dived for years without a problem and those who have had problems, will accept the opportunity to join Dr Richards' research project and provide useful facts on which to base diving medical opinion. At present much is based on assumptions which are not backed up by facts.

The meeting held at the Institute of Naval Medicine at Alverstoke in the UK in October 1990 has lead, as Des Gorman reports on page 74, to a new classification for decompression illnesses. We will have to accustom ourselves to talking of decompression illness, which includes both, rather than of decompression sickness (DCS) and cerebral arterial gas embolism (CAGE). The reasons are simple. They are often too difficult to distinguish and often occur together. The new scheme is a descriptive one based on the body system affected.

The long term sequelae of decompression illness are usually thought of as clear cut. Either the diver is left with paralyses or recovers completely. Our Personal Paper shows that some people make amazing recoveries. Dr Allan Sutherland's patient on the other hand, like those discussed by Dr Acott, shows a picture of long term mental changes which are severe enough to prevent him going back to his old job, or even working at all. The picture is similar to the results of traumatic head injury. At present there are no large series of such patients reported. Nor are there likely to be for these cases, though not uncommon, are not likely to return to their treating hyperbaric unit unless specifically asked to come. The only way diving medicine can go forward into this uncharted territory is to accumulate as many as possible of these case reports and study them. Any member caring for people who have long term mental changes, however slight, after a decompression accident should write the case, or cases, up for publication. It is clinical cases that point the way for changes in therapy (Dr Chapman-Smith's unfortunates who were denied surgery that might have restored their lost hearing are an example).

Incident reports are the equivalent of clinical cases and are just as important in establishing the pattern of diving problems. On pages 120 and 121 we reproduce the Diving Incident Monitoring Study (DIMS) form with a plea that it be photocopied and used to pass on anonymous information which can be used to assess the adequacy of the teaching given to divers and the equipment they use and to fromulate corrective strategies.

ORIGINAL ARTICLES

QUEENSLAND SCUBA DIVERS AND THEIR TABLES (CORRECTION)

Jeffery Wilks and Vincent O'Hagan

We regret that the table below did not appear with the above paper (SPUMS J 1991; 21(2): 11-14).

TABLE 1

CORRECT ANSWERS FOR THE TWO DIVE PROFILES: PERCENTAGE OF RESPONDENTS BY SEX AND TIME SINCE CERTIFICATION*

Group	Profile 1	Profile 2
Males	47	38
Females	39	34
Time since certification (months)		
0-12	50	44
13-24	45	39
25-36	37	27
37-48	52	42
>48	49	44

* Percentages are rounded to the nearest whole number.

LEARNING FROM THE MISHAPS OF OTHERS

John Lippmann

Towards the end of 1990 Coronial Inquiries were held into the deaths of four divers who died in Victorian waters in between October 1989 and April 1990. The Coroner decided to conduct the inquests together to determine any common threads and lessons to be learned, and to investigate whether government regulation of the diving industry is warranted. The hearings were held over 8 days and I had the opportunity to attend a number of the sessions.

I believe that there are a quite a few lessons to be learned from the misfortunes of these divers.

Case 1

The first fatality occurred during a Deep Diver Training Course. The victim was a 31 year old ex-RAN Clearance Diver who had started diving at 17 and who, apparently, had extensive diving experience, including a substantial amount of deep diving.

There were eight divers in the group (including the instructor) and they conducted the dive on the wreck of a paddle steamer, "The Coogee", situated off Port Phillip Heads in approximately 33 m of water. The divers were briefed to ascend after a pre-determined bottom time. When the bottom time had expired, the instructor ascended with five of the students, leaving the victim and his buddy unsupervised during the ascent. The instructor had thought it would be safe to do so as he believed the diver who died to be a more experienced diver than himself, which he probably was. The buddy began to ascend the anchor line but was pulled down by the victim, who appeared to be acting irrationally. When the victim removed his regulator from his mouth, the buddy handed him her primary regulator, transferring to her octopus, which she found more difficult to breathe from. After her mask flooded, the buddy became very anxious and indicated that they should ascend immediately. The victim then took the regulator from his mouth and began to ascend rapidly, without an air supply. He failed to reach the surface and his body was later located on the sea bed. When found, the victim had about 100 bar of air remaining in his tank and the recovery diver was able to breathe from the victim's regulator on the bottom. His weight-belt was still in place. Post-mortem examination revealed evidence of massive arterial gas embolism.

It is impossible to determine exactly what caused this accident and a number of scenarios have been suggested. It seems likely (because of the depth and some of the diver's behaviour) that nitrogen narcosis was a contributing factor.

Nitrogen narcosis affects all divers to some extent at depths approaching and beyond 30 m (some are affected at

far shallower depths from time to time), whether the divers are aware of it or not. Since most diving does not require a lot of focused, logical thought or fine movement, we may not notice the narcotic effects as we swim around at depth. Although reflexes and thought processes may be slowed down, most diving situations do not require an immediate, rapid, rational assessment and co-ordinated reaction. However, if a problem develops, we need to think and act swiftly, and this is when the effects of narcosis, previously unnoticed, may prove debilitating. We may act irrationally and clumsily and are more likely to panic, so endangering ourselves or our buddies, or both.

Experienced divers, such as this victim, are still susceptible to narcosis, especially if they have not dived deeply recently, and, at times, experienced divers may act irrationally and may panic under certain adverse circumstances. Experienced divers, just as any other divers, must be vigilant for the effects of narcosis on deeper dives and must monitor their buddy, and, in turn, be monitored by their buddy.

When the victim's regulator and cylinder were tested a number of interesting observations were made. The cylinder valve was a "J valve" (i.e. fitted with a reserve mechanism) and when the reserve lever was in the "on" position the air flow was greatly reduced, even at relatively high cylinder pressures. Unfortunately, no record was kept of what position the valve was in when the diver was found so it is not clear if this potential problem did, in fact, contribute to the accident. In addition, the diver had recently fitted a high-flow (Oceanic) second stage to what was possibly an incompatible first stage (Apollo). The police tests indicated that it was likely that a diver, breathing rapidly at depth, could out-breathe the regulator.

The following is a possible explanation for the accident:

The victim may initially have had difficulty getting enough air from his regulator at depth. After transferring to his buddy's regulator he appears to have had problems using his buddy's regulator. These problems are likely to have been exacerbated by nitrogen narcosis which would itself be exacerbated by carbon dioxide retention, caused by a hard-breathing regulator, and by anxiety. It appears that the victim then decided to "free ascend" (which he would have been trained to do in the Navy). He failed to exhale appropriately and, consequently, suffered an arterial gas embolism, losing consciousness underwater.

Lessons from Case 1

First, all divers, especially those who dive beyond about 24 m, must familiarize themselves with the manifestations and management of nitrogen narcosis, and be vigilant for signs of it in themselves and their buddies during deeper dives.

J valve reserve mechanisms can, and do, malfunction. They are best removed completely and replaced with a blanking plug. Taping them down in the open position does not guarantee that the mechanism will always remain deactivated. If a diver wishes to keep an operational "J valve", he or she should ensure that it is inspected and serviced regularly by an appropriately experienced service technician.

Certain second stage regulators may be incompatible with certain first stage regulators. A diver should ensure that his equipment is compatible and functions adequately.

Many octopus regulators can be difficult to breathe from, especially in deep water when the cylinder pressure is relatively low. Divers should ensure that they have an octopus that is properly maintained and is capable of supplying air to a rapidly breathing diver at the depths to which the diver dives.

An instructor should endeavour to keep all his or her students in sight, and within relatively easy reach, throughout any training dive. This is important during deep dives when narcosis may affect a diver's behaviour, an air supply problem is more likely and ascent more difficult. It is often very difficult for an instructor to monitor a large group of divers and, consequently, instructors should think carefully about how many divers they can adequately monitor during a particular training dive. Ratios should be chosen with diver safety, rather than commercial pressures, as the primary consideration.

A diver, who finds himself in a situation where he fears he may become unconscious during ascent, should either make himself positively buoyant or, alternatively, remove his weight-belt and hold it in his hand and away from his body. If he becomes unconscious the belt will fall away and the diver should rise to the surface. A diver on the surface generally has a far greater chance of being successfully rescued than one on the bottom.

When dives deeper than about 30 m are conducted it is often a good idea to attach a full cylinder (capacity of at least 400 litres) with a properly functioning regulator to the bottom of the ascent line. It should be attached by an easily removable clip. A diver who is low on air on the bottom can remove the tank and use it on the way to the surface.

Case 2

Another fatality occurred near "The Coogee". This victim was a 25 year old man who had done his initial dive course in 1983 and had been diving regularly over the past

few years. He, too, was diving from a commercial dive charter vessel.

The sea conditions were relatively poor. According to the buddy, the divers descended to the bottom (at 33 m) and the victim swam away quickly, making it difficult for the buddy to keep up with him. Apparently, the victim then turned to his buddy and signalled that he wanted to buddy-breathe. Since the buddy did not have an octopus, the divers decided to share the one regulator. It was reported that they exchanged the regulator for about four cycles without leaving the bottom. As they were incorrectly positioned for buddy-breathing, the buddy was passed the regulator upside down, causing him to inhale water. They began to ascend slowly, too slowly for the buddy's liking, so he inflated his buoyancy compensator (BC) and very rapidly ascended to the surface without his regulator in his mouth, leaving the victim behind. The buddy arrived relatively safely at the surface, but the victim never reached the surface. When his body was later recovered from the sea-bed there was damage to the his mask and face. His weight-belt was still in place. About 130 bar of air was left in his cylinder. Post-mortem examination showed evidence of arterial gas embolism but death was recorded as due to drowning.

When the victim's gear was tested a number of observations were made. The cylinder was only turned on approximately one-quarter of the way. This may have been sufficient to supply air at the surface but would have made it difficult to get adequate air at depth, especially when under exertion or stress. In addition, the line pressure in his regulator was far lower than it should have been, and this, too, would have reduced the air supply, as would have the dirty sintered filter. It appears that the victim serviced his own gear but had inadequate training to do so safely. He had attended an Equipment Specialist Course, but the course was not designed to teach divers how to service their regulators. His BC filled only slowly at the surface and very slowly at depth.

It is probable that the victim could not get enough air from his own regulator on the bottom as the line pressure was too low, the filter was dirty and the valve inadequately turned on. The divers failed to buddy-breathe successfully due to lack of practice (by both himself and his buddy) and the effects of nitrogen narcosis. When the buddy inflated his BC and rapidly ascended, the victim may have been forced to rise without exhaling adequately and suffered an arterial gas embolism. At some stage he lost consciousness, sank to the bottom and drowned.

Lessons from Case 2

Divers should ensure that their equipment (i.e. regulator(s), cylinder and valve, BC and gauges) are inspected/serviced at appropriate intervals by by an adequately experienced service technician.

A diver must ensure that his or her cylinder valve is turned on adequately. Divers are generally taught to open the valve fully and then turn it back some amount (often one quarter or half a turn, or thereabouts) so that if, knocked, the valve will not jam on. However, with some valves, turning back half a turn may reduce the air flow significantly. In addition, on a number occasions I have seen well-meaning divers inadvertently turn off their buddy's valve and turn it on a quarter turn, creating a very dangerous situation. I believe that it is generally simpler and safer to teach divers to turn the valve on fully (valves rarely jam) and then check the air flow by watching the contents gauge while purging the regulator. If the needle on the gauge fluctuates, there is probably too little air getting through.

Buddy-breathing is often unsuccessful as it is a technique that needs constant practice by both participants and requires a great degree of presence of mind in an emergency. I believe that all divers should carry a properly functioning "octopus regulator" (or similar device) and should ensure that they can use it properly. It should be positioned where it is rapidly accessible to its wearer (not tucked away in a pocket!).

Case 3

The fatalities included one snorkeller. The victim was an Assistant Scuba Instructor/Snorkel Instructor who was a very experienced and enthusiastic diver. He was also an asthmatic.

The victim was with a group of ten people, mainly diving instructors. They had just done a pre-Christmas "pleasure dive" and decided to anchor the boat in shallow water and snorkel for crayfish. The snorkellers went off individually, and, since they were all very experienced, there was no pairing off and on-one was assigned to be a look-out. It was Christmas Eve and the mood was very relaxed.

When all the other divers had returned to the boat and the victim could not be seen anywhere, the group became worried and instigated a search. The victim was found a number of hours later on the bottom in about 3 m of water. He was lying in kelp but was not tangled in it. The recovery diver released the victim's weight-belt (which was still in place) and brought the body to the surface. Post mortem examination showed nothing other than signs of drowning. It is not known whether asthma played any part in this misadventure.

Since no-one witnessed this diver's difficulties, one cannot be sure what caused this fatality. However, many of

us who knew the victim feel that it is likely that he hyperventilated before a breath-hold dive. Among other aspects, he enjoyed the risks of diving and at times was known to push safety to the limits. Most divers are taught the dangers of hyperventilating before a breath-hold dive (i.e. the possibility of losing consciousness underwater or shortly after ascending), but some divers still hyperventilate to increase their breath-hold time. The likelihood of a post-hyperventilation blackout varies from person to person and, within an individual, from one time to another.

The victim, who was an ardent crayfish hunter, may have spotted a crayfish and hyperventilated to extend his dive time enough to catch the creature. It appears that the victim was too heavily weighted, so, when he lost consciousness he would have sunk to the bottom, rather than rising to the top as he may have if he had been positively buoyant. Since no-one witnessed his failure to surface, drowning was inevitable.

Lessons from Case 3

Hyperventilating before a breath-hold dive can, and does, cause some snorkellers to become unconscious in the water, often leading to drowning. Some instructors teach that it is safe to hyperventilate a few breaths before a duck-dive, but I believe this to be foolhardy. As previously mentioned, there is a lot of variation and, what may have proved safe on one occasion may not be safe on another. Some snorkellers have been known to blackout after a very long breath-hold dive, even without hyperventilating. A number of highly experienced snorkellers have suffered post-hyperventilation blackout, and many of them subsequently drowned. Since regular breath-hold divers can learn to endure high carbon dioxide levels and suppress their urge to breathe, they have an increased risk of becoming unconscious from the lack of oxygen, caused by delaying inspiration.

Snorkellers should never be negatively buoyant. They should be slightly positively buoyant so that they are likely to rise to the surface (or remain on the surface) if they become unconscious.

Snorkellers should, where possible, avoid diving alone. The buddy system should be encouraged for many snorkeling activities.

Case 4

The final victim was a 51 year-old man who had very limited diving experience in Queensland waters, and had completed a scuba diving course, during a cruise on board the Fairstar, a few months before his death. He had been medically examined prior to his dive course but it appears likely that the examining doctor lacked the appropriate training in diving medicine. His fitness and health were quite poor and he was taking a medication that affects the heart rate. He had recently bought new diving equipment, including a 7 mm wetsuit, and this was his first dive in relatively cold Victorian waters.

The victim was diving from a commercial dive charter vessel at a relatively safe, although current prone, site, with a maximum depth of around 12 m. After entering the water and signalling "OK" to his buddy, the victim deflated his BC but only descended slightly and began to drift away from his buddy and the boat. When he signalled he was in trouble, the skipper raised the anchor and brought the boat alongside the diver. A rope was thrown to him and, although it landed over his shoulder and in front of him, he did not take it. The victim still had his regulator in his mouth and bubbles appeared to be coming from it. The rope was thrown again but the deceased drifted away and sank. Those present thought he had begun his dive, alone and in the current, rather than in the sheltered area. When the bubbles could not be seen the skipper became concerned and organised a search. The deceased's body was found some hours later. Post-mortem examination found that death was caused by drowning.

The Coroner suggested that a number of factors may have contributed to this diver's death. It was his first dive in relatively cold water and, probably, with a full 7 mm wetsuit. The cold water, the wetsuit, anxiety and the medication he was taking could have made breathing difficult, clouded his consciousness and, eventually, may have caused his heart to stop beating effectively, leading to unconsciousness and subsequent drowning.

Lessons from Case 4

This fatality is a reminder of the importance of ensuring that we are healthy enough and fit enough to dive safely. Although most divers are now encouraged to have a medical examination before taking up the sport, unless the examining doctor has knowledge of underwater medicine he or she may not be fully aware of the dangers certain medical conditions may pose to a diver. Many hyperbaric experts believe that it is important for divers to be examined by a doctor who is appropriately trained in diving medicine, to determine whether they have any conditions that may predispose them to a serious diving accident. As our health changes from time to time, it is wise to be re-examined after any significant change in health status. Some diving certification agencies recommend that divers be examined every five years until the age of thirty, every three years from thirty to fifty and annually thereafter.

Since diving has inherent risks it is inevitable that some fatalities occur. However, if we consider the number

of divers and thedives that are done each year, the fatality rate seems to be very low. We can never completely guarantee our safety during a dive, but we can certainly minimise the chances of a problem by ensuring we are healthy, fit and prepared enough to dive, that we have suitable and serviceable equipment, that we are adequately trained and experienced to do the particular dive, and that we use our common sense, and do not push the limits of safety.

The above article has been slightly edited from the form in which it will appear in a future issue of "Sportdiving in Australia and the South Pacific" and is reprinted here by permission of the author and of the publisher.

We look forward to publishing a lawyer's view of these inquests.

John Lippmann is the author of "The DES Emergency Handbook", "The Essentials of Deep Diving" and "Deeper Into Diving", all of which are available from J.L.Publications, P.O. Box 381, Carnegie, Victoria 3163, Australia.

ASTHMA AND DIVING.
Some Observations and Thoughts*

Carl Edmonds

People keep asking me for reprints of papers that I wrote on asthma years ago, in such literary papers as the SPUMS Journal, Pressure Newsletter, Undercurrent, etc. I do not keep copies of the past. In the hope of deflecting further requests, I present here selected excerpts from the 3rd edition of Diving and Subaquatic Medicine, which is in press and which will be distributed by the publisher, Butterworths, possibly in 1991-2. Requests for complimentary copies should be sent to them, not to me.

Asthma and diving deaths.

Only 1 % of American divers have a history of asthma, as judged from the 1988 DAN diver survey.[1] The figure is probably less in Australia, where medical questionnaires and examinations are required before diving. However at least 9 % of the deaths in Australian and New Zealand recreational divers[2] were in asthmatics and in at least 8% it was a major contributing factor.

* Edited excerpts from *Diving and Subaquatic Medicine,* 3rd Edition Butterworths (in press)

Most of these deaths were in clinically mild asthmatics who are otherwise physically fit young men.

The possible trigger factors for asthma provocation in scuba diving are;

1 Exertion (from overweighting, equipment drag, swimming against tides etc.),

2 Inhalation of cold, dry air (adiabatic expansion of dehumidified compressed air),

3 Hypertonic saline inhalation (bubbling or leaking regulators),

4 Breathing against a resistance (increased gas density, regulator problems, low air supply).

Many of these stresses are used clinically to initiate asthma as diagnostic provocation tests, and so the problems with this disorder are understandable. In a number of cases the diver was returning to obtain a salbutamol (Ventolin) spray; in others it had been used immediately before the dive.

Asthmatics, even more than others, had multiple contributions to death. The relative frequency of a compromised air supply, salt water aspiration, panic and fatigue, prior to drowning, was evident from the statistics

Asthma and diving accidents

Most experienced diving medical physicians are appalled at the though to asthmatics diving. This attitude stems from a number of places. They include:

1 Involvement with asthmatics who died;

2 The catastrophic histories from those who survived;

3 Differential diagnostic difficulties with asthmatics who have near-drowning and possibly also pulmonary barotrauma;

4 Therapeutic complexities in these cases, both regarding depth and oxygen exposures, and drug complications;

5 Training from their teachers, whose experience often

TABLE 1

ASTHMA DEATHS FROM 100 RECREATIONAL DIVING FATALITIES IN AUSTRALIA AND NEW ZEALAND

Autopsy cause of death	
drowning	7
pulmonary barotrauma	2
Other medical contributions	
salt water aspiration	5
fatigue and/or panic	5
Technique problems	
Compromised air supply	6

is based on earlier times, when there was no diver medical selection.

Most of us have seen too many asthmatics who, because of a commendable desire to achieve normality, are affronted by advice that they should not scuba dive. They tend to be very fit, somewhat macho, males with a type A personality. They, correctly, claim that their asthma is mild, easily correctable, and not a restriction in other sports. In fact, any clinically severe asthmatic is most unlikely to ever intend to dive.

Often they extrapolate their techniques of pre-exercise medication to diving, without realising that this will increase the likelihood of the sudden death syndrome, which had now become one of the commonest causes of recreational diving deaths. In many cases the diver was returning to obtain a salbutamol (Ventolin) spray, when the accident or death occurred, in others it had been used immediately before the dive. Some believed that the spray overcomes all airway restriction, not realising that the pharmacological effect is patchy and competes with the provoking factors.

The asthma provocation factors in scuba diving are mentioned above. Especially serious incidents tend to occur when more than one factor is present. These provoking factors, which lead to dyspnoea, fatigue, difficulties with the regulator or gas supply, panic emergency ascents, salt water aspiration, near-drowning or drowning, pulmonary barotrauma, etc, are rarely seen with free diving or recreational swimming, except in extreme circumstances.

Case Reports

These were selected from our own files, except where otherwise stated, to illustrate the variety of presentations. They are not from the ANZ death statistics referred to above.

Case 1

A, aged 25, was a very fit, mildly asthmatic, sportsman. He had been diving for four months when he went to 18 m for 20 minutes. Without an obvious reason, he performed a rapid ascent, developing dyspnoea and confusion on the surface and left sided paralysis within a few minutes. He was taken by helicopter to the Royal Australian Navy recompression chamber (RANRCC). He was initially compressed to 18 metres on oxygen, but as he did not regain consciousness he was then taken to 50 m.

After a three-day vigil, in which the patient was subjected to various procedures in an attempt to surface him, he died, still under pressure.

During that time he was treated conscientiously for his asthma, which was evident on auscultation, and for CAGE. He was given steroids and anticonvulsants (for his repeated epileptic episodes), measures to counter possible cerebral and pulmonary oedema, and to maintain his electrolyte and pH levels.

The autopsy revealed mild cerebral oedema, congestion of the meningeal vessels and ischaemic cell damage in the right frontal lobe and the the right thalamus. There was a tear on the posterior section of the upper lobe of the right lung, with intra-alveolar haemorrhages and rupture of alveolar septae. The lung basal membranes were thickened and muscles showed hypertrophy, consistent with asthma.

Provisional diagnosis:

Asthma, pulmonary barotrauma, CAGE.

From the Royal Australian Navy School of Underwater Medicine

Case 2

B, aged 33, had been a qualified diver for four years, despite being a known, but very mild, asthmatic. He was classified as fit by a doctor who alleged experience in diving medicine. The doctor also gave a script for salbutamol, and advised him to take it prior to diving. He followed this advice. He even had a pocket included in his wet suit to hold the inhaler.

He descended to 9 m for 20 minutes, then did an ascent to get his bearings. On returning to his companion, he appeared distressed and then made a further rapid ascent to the surface. There he appeared to be confused and removed the regulator from his mouth. He inhaled some sea water and then lost consciousness and went into a convulsion.

He was rescued by his companion, and within 30 minutes reached the RANRCC, by helicopter. He was comatose with brain stem spasms and with a very inadequate air entry, bilaterally. He was compressed to 18 m on oxygen.

Despite endotracheal intubation and 100% oxygen, at 18 metres, with positive pressure respiration, the PaO_2 level remained at 50-70 Torr. The $PaCO_2$ levels were usually above 100 Torr and the pH remained below 7.0.

Mainly because of the death of an almost identical asthmatic diver, just previously, after a descent to a much greater depth, it was decided to surface this patient over a period of approximately five hours, while attempting to maintain as high an oxygen partial pressure as possible.

The problem was in the combination of diagnoses, including cerebral gas embolism (the initial incident), asthma

(as detected by the significant bronchospasm) and drowning (caused during the attempted surfacing and rescue of the patient).

Going deeper, to overcome the effects of the air embolism, would be complicated by prejudicing his PaO_2 level. The greater depth and increased density of the gases would probably interfere with adequate ventilation, CO_2 exchange and cause acidosis.

Aminophylline could cause arterialisation of pulmonary air emboli. The coincidental hypothermia (33-35 °C) was not considered a definite problem, and might even be advantageous, if it was not for the effect sympathomimetics, required for the asthma, could have on cardiac arrhythmias. Steroids were given for the rather indefinite, but multiple reasons, as given above (asthma, cerebral damage, drowning, etc.).

Initially the chest X-ray verified gross pulmonary oedema, consistent with the combined effects of asthma and drowning. Subsequent chest X-rays revealed a persistent right lower lobe opacity, clearing up over the next month.

With attention to the respiratory status, the brain damage, fluid and electrolyte status, the patient gradually improved over the next few weeks and he regained consciousness. The result was a severely brain-damaged young man continually incapacitated by myoclonic spasms, which were almost certainly post-hypoxic but possibly contributed to by CAGE. There was a residual dysarthria, a left hemiparaesis, an ataxic gait and myoclonic jerks. The EEG was consistent with hypoxia and the CT scan was normal.

Provisional Diagnosis.

Asthma, CAGE, near-drowning.

Case 3

C, aged 43, was a very experienced diver who previously had asthma as a child, and who still had high pitched rhonchi on auscultation during hyperventilation.

A very eminent respiratory physician informed him that his lungs had quite adequate function for scuba diving. This advice was refuted by members of the Diving Medical Centre but academic brilliance won out.

Whilst exploring a wreck, at a depth of 27 to 18 m, he suddenly became aware, as he floated up over the deck, of a pain in the left side of his chest. He then attempted to ascend. The pain became worse as he ascended. He slowed down and the pain decreased. He took over half an hour to reach the surface. During this time there was a continual pain in the chest, aggravated if he tried to ascend rapidly.

With extreme courage, and commendable control over his breathing gas consumption, despite the terrifying circumstances, he did reach the surface, although in great discomfort. He was then given oxygen and transferred to hospital. The clinical and X-ray evidence verified the presumptive diagnosis of left pneumothorax, and a thoracentesis was performed.

He returned to the respiratory physician, to be reassured that it was unlikely to happen again. The Diving Medical Centre physicians, assured him that not only would it happen again but that, with the lung damage and the treatment received, it was more likely to happen again and that it should not have been allowed to happen in the first place. He decided, this time, to take our advice.

Provisional Diagnosis.

Asthma, pneumothorax with minimal provocation.

Case 4

D, aged 20, had been certified fit to dive despite an asthma history. Prior to the dive there were no symptoms, but he still took a salbutamol inhalation.

In his first deep water dive, after spending 8 minutes at 30 m, he took 23 minutes to reach 15 m. A burning pain in his chest then caused him to make a rapid ascent. He was pulled out semi-conscious and apnoeic. He had four grand mal seizures and was given oxygen on examination. There were no neurological defects other than disorientation, After 6 hours, during which time he had another three seizures, he was recompressed to 18 m on oxygen and treated with anticonvulsants. There was no evidence of a pneumothorax, and he was eventually treated on an air table at 50 m having continued to convulse while on O_2 at 18 m. He survived, but has subsequently stopped scuba diving.

Provisional Diagnosis:

Asthma, pulmonary barotrauma, CAGE,

Summarized from SPUMS Journal, reported by of Dr. David Clinton-Baker.

Case 5

E, age 23, was a very fit and courageous athlete, who had mild asthma and was advised against scuba diving. Unfortunately his father, who was a professor of medicine, succumbed to family pressure and signed a “fit for diving” certificate.

This patient suffered two episodes of a very similar nature. In neither case had he had any evidence of active asthma prior to the dive, and in the second episode he had actually taken a salbutamol spray before the dive. These dives were in a similar area to depths less than 10 m. After 20 to 30 minutes he had developed dyspnoea and attempted to return to shore. On the first occasion he had informed his buddy that he was returning to shore to get another salbutamol

TABLE 2
ASTHMA ASSESSMENT PROTOCOL

History of asthma over the last five years.	FAIL
Use of bronchodilators over the last five years.	FAIL
High pitched respiratory rhonchi or other respiratory abnormalities.	FAIL
High pitched expiratory rhonchi, on hyperventilation.	FAIL
High pitched expiratory rhonchi on hyperventilation ten minutes after a 5min @ 900 KPM/min exercise stress.	FAIL
FEV1/VC of <75 % of predicted value.	FAIL
Expiratory flow rates of < 60% of predicted value. (Basic spirometry; FEF 25,50, 75, MMEF etc.)	FAIL
Asthma provocation producing >10% reduction of expiratory flow rates FEV 1 or PEFR) after both conventional histamine and hypertonic saline provocation, preferably while breathing dehumidified cold air.	FAIL

If all the above are negative or clear, limited diving may be permitted to a maximum depth of 18 metres without any free ascent practice.

spray but he appeared to panic and inhaled sea water. He was then rescued in a comatose state and eventually recovered after helicopter transfer to a major hospital.

The second episode was of a very similar nature, except that he did not recover. The autopsy revealed evidence of drowning, with mild asthma.

Provisional Diagnosis:
Asthma, panic, near drowning and drowning.

Case 6

F, age 20, was a fit young diver who carried out 30 scuba dives to a maximum of 39 m, without incident, before being certified as fit for diving by an experienced diving physician. There was a past history of asthma for which he had used steroid inhalers. On examination there was no evidence of bronchospasm and the FEV1/VC was $3.9/4.5_1$, without bronchodilators. The chest X-ray was normal. He was advised that he would be medically fit to dive providing he was free of asthma and that he taken an inhalation of Berotec prior to each dive.

While undertaking in-water rescue and resuscitation exercises, to a maximum of 4.5 m he developed dyspnoea on the surface. He informed the instructor that he was suffering from asthma and was towed 30 m back to shore. By then he was cyanosed with wheezing on inspiration and expiration. He then lost consciousness and required expired air resuscitation (by two experienced internists). He suffered a grand mal seizure and then gradually improved following oxygen inhalation. He responded to treatment of his asthma, over the next few days, using aminophylline.

There was no evidence of CAGE, and the seizure was considered to be due to cerebral hypoxia. Later a history of an asthmatic episode four days previously was elicited. It was presumed that the asthma was triggered by the aspiration of sea water, exertion and cold exposure.

Provisional Diagnosis:
Asthma, near drowning

Case 7

"I have extremely mild asthma, which manifests perhaps once every three years for a brief time during a respiratory tract infection. As I did not encounter any asthmatic symptoms during strenuous high altitude mountaineering I thought it would be reasonable to try scuba diving. I learnt to dive in a warm shallow swimming pool and experienced no difficulties during this or my first sea dive. During my first deep sea dive, however, I had an extremely severe and sudden attack of bronchospasm at a depth of 30 m. I barely made it to the surface, where my obvious distress and lack of speech caused my partner to inflate my life jacket, thus compromising my respiration further. It was a frightening experience and I have not dived since."

J .J .Martindale. Summarised from a letter in reply, in the BMJ.

Provisional Diagnosis
Asthma, panic.

Protocol for assessing potential asthmatics

The protocol in Table 2 has been of value to us for the assessment of diving candidates with a history of asthma and a normal chest X-ray. If all the findings are negative or

clear, limited diving may be permitted to a maximum depth of 18 metres without any free ascent practice.

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A NEW CLASSIFICATION FOR THE DECOMPRESSION ILLNESSES.
Report on a workshop held at the Institute of Naval Medicine, Alverstoke, United Kingdom, October 1990

Des Gorman

Recently the Royal Navy Institute of Naval Medicine (INM) sponsored a 2 day workshop at the Institute to develop a new classification for the decompression illnesses. This was prompted by an attempt at organising a multi-centre trial of lignocaine in cerebral arterial gas embolism (CAGE), which foundered when diagnostic criteria for CAGE could not be agreed. Funding for the workshop was provided by INM and 35 delegates were invited and attended; including the author, Carl Edmonds, David Elliott, James Francis, Tom Shields, Ed Thalmann, Ed Flynn, Drew Dutka, Ramsay Pearson, Lindsay Symon, David Dennison, Richard Moon, Maurice Cross, Ian Calder, Hans Ornahagen and Yehuda Melamed. The proceedings will be published by the Undersea and Hyperbaric Medicine Society.

The existing classification.

Before the Workshop the decompression illnesses were conventionally divided into CAGE and decompression sickness (DCS). DCS was further divided in types I (mild) and II (serious) in a system proposed over 30 years ago for caisson work.[1] The workshop participants agreed that although sudden loss of consciousness in a scuba diving candidate on surfacing in a swimming pool was almost certainly CAGE and that left knee pain in a saturation diver developing 6 hours after reaching the surface was similarly certain to be DCS, between these two extremes differentiation was often impossible.

Furthermore it was agreed that:

a CAGE can present before reaching the surface;

b almost all cases of cerebral DCS have symptoms within 20 minutes of surfacing;

c many cases of cerebral DCS were likely to be due to arterialisation of venous bubbles and hence that DCS often initiated CAGE;

d arterial emboli could either precipitate DCS or occur concurrently with DCS (the so-called type III DCS [2]);

e most cases of CAGE did not have any evidence of lung damage;

f in submarine escapees de-novo formation of bubbles in arteries could not be completely excluded; and,

g while most cases of CAGE showed some spontaneous recovery many were static or progressive.

Many delegates reported that attempts at retrospective analysis of case histories had resulted in very low concordance between observers in the diagnosis of either

CAGE or DCS. Also, the recent decision of the United States Navy to treat CAGE initially at 2.8 bar breathing 100% oxygen meant that most of those attending had no incentive to make the distinction as treatment regimens were essentially common. This is particularly true given the move away from a significant head-down posture for CAGE sufferers.

Similarly, the type I and type II DCS classification was considered unsatisfactory because:

a if left untreated at least 30% of type I DCS cases developed overt evidence of neurological involvement;

b the frequency of long-term personality, psychological and neurological sequelae was almost as high in divers with a history of type I DCS as in those with type II DCS;

c type II DCS could indicate anything between a diver with paraesthesia in the left finger and either an unconscious diver or a diver with intractable hypotension and shock;

d in diving operations, technicians and divers (and most doctors) are unable to identify subtle neurological signs and hence make a diagnosis of type II DCS; and,

e much of the pain in DCS was likely to be referred from the nervous system.

It was accepted unanimously then that the existing classification needed to be changed and that for the reasons given above neither an aetiological nor an organ-system classification was achievable. The latter would be made even more difficult by the multi-focal nature of the decompression illnesses.

The consensus then was that:

a no attempt should be made to distinguish CAGE from DCS; and,

b a clinical descriptive classification should be developed.

A clinical descriptive classification of the decompression disorders

The term decompression illness was proposed and accepted to include both the previous DCS and CAGE categories and to demonstrate that no distinction was being made.

It was agreed that this term should be prefaced firstly by an evolutionary term (static, resolving, relapsing, progressive etc) and secondly by an organ system term (these being the symptomatic organ systems). For example: a diver who collapsed on surfacing, was found to be unconscious and then recovered would have "resolving neurological decompression illness"; a diver who had increasing shoulder pain would have "progressive musculoskeletal decompression illness"; and, a diver with unchanging shortness of breath and paraplegia would have "static pulmonary and neurological decompression illness". The last case illustrates that no attempt is made at putting symptoms into a hierarchy.

The overall classification of decompression disorders would then be:

a **Barotrauma**
- ENT
- Pulmonary (radiologically or clinically apparent pneumothorax, mediastinal and sub-cutaneous emphysema)
- Others

b **Decompression illness**

The lignocaine trial.

It now becomes possible to perform the planned CAGE-Lignocaine trial by identifying the applicable clinical syndromes (e.g. only progressive neurological decompression illness cases).

Other conclusions

In addition to agreeing to a trial of this new classification, the participants conceded the need for a common database for diving accidents. The composition of this database will form the basis of a subsequent workshop.

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LATE SEQUELAE OF DECOMPRESSION SICKNESS
A case report

Allan Sutherland

This is the story of T, a 41-year-old male diver who has suffered, and continues to suffer, significant psychological and physical disabilities after two episodes of decompression sickness in July 1987 and February 1988. The medical assessment of this man has been difficult, with many reports conflicting, resulting in delays with compensation.

He suffered his first diving accident in July 1987 during his second dive of the day. This dive was to greater than 30 m looking for crayfish. He became wedged under a rock and was only able to free himself by removing his backpack buoyancy compensator and using his octopus regulator. Separated from his gear, but holding it, he surfaced rapidly.

By the US Navy tables he exceeded the no-stops dive limit by at least 19 minutes. He was therefore a candidate for both decompression sickness, by virtue of the length of his dive, and air embolus, because of his rapid ascent.

On the surface he felt "funny" and noticed tingling of his lips, fingers and toes. Subsequently, 3 hours later, he developed pain in his left shoulder and right hip. He was unable to pass urine and had an unsteady gait.

On examination, both the left shoulder and right hip were painful on all movements, with associated weakness around each of these joints. He had hyperaesthesia of his left arm and loss of sensation to pin prick over the dorsum of his right foot.

He was treated with an extended US Navy table 6, receiving a total of 12 hyperbaric oxygen treatments.

Two and a half weeks after this accident a neurologist reported that T had a slight pyramidal weakness on the right side and extensor plantar responses on that side, with pin prick and position sense impaired in the right leg. He was unsteady standing on either foot, even with his eyes open. The neurologist's report included "I agree that this man has had decompression sickness affecting the cerebral hemispheres and spinal cord. I would expect him to make a complete recovery from these problems within 3 months."

Contrary to medical advice, T resumed diving and sustained a further diving accident in February 1988. After a late night, and excess alcohol, T did a strenuous dive for about an hour at 18 m. He became muddled, and developed pains in the hips and surfaced. He saw flashes of light at about 5 m but he remembers very little until he was put in the recompression chamber.

T's worst symptom continues to be pain in the right hip. He has a balance problem and does not seem to empty his bladder properly. He claims that his concentration is not as good as it should be. He tends to drop things but has not noted any specific impairment of his hands.

On examination recently there was slowness in performing rapid alternating movements of his arms, with possibly some slight impairment of position sense. He was mildly ataxic, walking with his feet a few inches apart. There was impairment of position sense bilaterally, with hyperaesthesia in the right leg. The tendon reflexes were reduced and the plantar responses were flexor.

Now the neurologist comments "This episode of bends seems to have produced mainly spinal cord problems which are relative minor and should recover completely. From a neurological point of view, I consider that he will, again, make a complete recovery".

There are now, in all, 16 reports available on T. Many are conflicting in their content and clearly demonstrate the great difficulty the medical profession has in appropriately assessing the long-term effects of decompression sickness, especially the personality changes and soft neurological deficits.

In an attempt to help this man two neuro-psychological assessments were done. The first was performed in November 1988 and, nineteen months later, the second in July 1990. The test performed were Block Design, NART, PASAT, Picture Completion (WAIS-R), Quick Test, Key Complex Figure, Stroop Test, Trail Making Test, Visual Sequential Memory, Visual Reaction Times (two forms), Wechsler Memory Scale (Form II) and Word Fluency.

The final neuro-psychological report states that T was having a lot of difficulty concentrating and was unable to cope with any situation where things were happening fast, including situations were there was a lot of noise, where several people were talking at once. This difficulty affects his family and social relationships and has implications for his ability to work. When his children are noisy, or racing around, he gets very irritated and cannot handle it. Similarly, in many social situations, he is unable to follow conversations and just switches off. Consequently there are many social contacts that he now avoids.

When he wants to write "It all gets jumbled up". Although he is able to drive a car, he does not do this often as he has to put so much effort into it and after driving feels exhausted. Both there difficulties are consistent with impaired ability to concentrate and a slowing of the thinking

process.

Difficulties of a more physical nature included difficulty sleeping more than a few hours a night although he feels worn out, difficulties with sex, which include variable difficulty in gaining an erection, also pain and lack of feeling during intercourse, and a reduced tolerance to alcohol.

He is learning to accept his intense frustration and anger, adapting to frustrations by avoiding them. T feels anti-depressants have been helpful, not just to improve the mood state, but they also permit him to tolerate his disability and its frustrations. However his wife thought they made him more aggressive.

This man has been left with the kind of cognitive impairments that frequently follow other forms of diffuse brain damage. The pattern of his neuro-psychological profile is strongly suggestive that he has organic dysfunction of the brain. This continues to have a profound impact on his family relationships and his ability to work.

Discussion

After some 3 years, and many conflicting reports, T is still awaiting final permanent disability settlement payments. He continues to have a poor attention span, impaired concentration, reduced short-term memory, depressed mood, labile emotions, language difficulties, impaired balance, weakness of his left shoulder muscles, occasional paraesthesiae in the right leg, episodic diarrhoea and urinary hesitancy.

Despite these crippling problems we have optimistic neurological reports in conflict with the neuro-psychological assessments which are more in keeping with our diving medical assessments. Others have noted that "The cerebral recovery from DCS and AGE is more refractory than previously thought".[1] T is not alone, he is one of 8 divers who were unable to return to their previous employment, because of persisting disorders of their higher intellectual function and mood state, after hyperbaric treatment at HMNZS PHILOMEL for a diving accident.[2] There were 30 patients in the series reported. These 8 patients were assessed as impaired using the criterion of return to usual employment. The patients were invited to make self-assessments. Assessment was also made by the spouse and by the family practitioner. In addition psychometric testing was performed at the Post-Concussion Clinic, Auckland Hospital.

All these assessments gave evidence of disturbed higher intellectual function and mood state, with considerable variability of the symptoms. Although the assessments have a large subjective component, and the report is brief, the conclusion is statistically significant (8/30) and the patients' disabilities are very real. The paper's conclusion was "That the cerebral effects of sport diving accident cases are refractory when assessed by family observation, G.P. observation, and psychometric testing".

This case clearly demonstrates the difficulties in quantifying the late sequelae of decompression sickness and achieving appropriate compensation payments.

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FREMANTLE HOSPITAL HYPERBARIC MEDICINE UNIT THE FIRST YEAR

Harry Oxer and David Davies

Introduction

After 12 years planning, and annual submissions to the Government, the Hyperbaric Unit at Fremantle Hospital was opened for business on November 27th, 1989. Construction was directed by Hyox of Scotland but most of the work was subcontracted to local West Australian companies.

The Unit is located in a former laundry and has facilities for consultation and wound care in addition to the administrative and treatment areas. Its effectiveness was justified by achieving the planned operational goals within six weeks rather than the expected six months.

The treatment unit consists of two hyperbaric chambers connected to a separate entrance lock which has provision to lock-on a transfer under-pressure module (Figure 1).

"Mildred", as the main treatment chamber is known, is a vertical cylinder with an internal diameter of 3 m and is 3 m high. It has a large rectangular door through which patients can walk or be wheeled on trolleys. The door is large enough to allow a Drager Duocom transportable chamber to

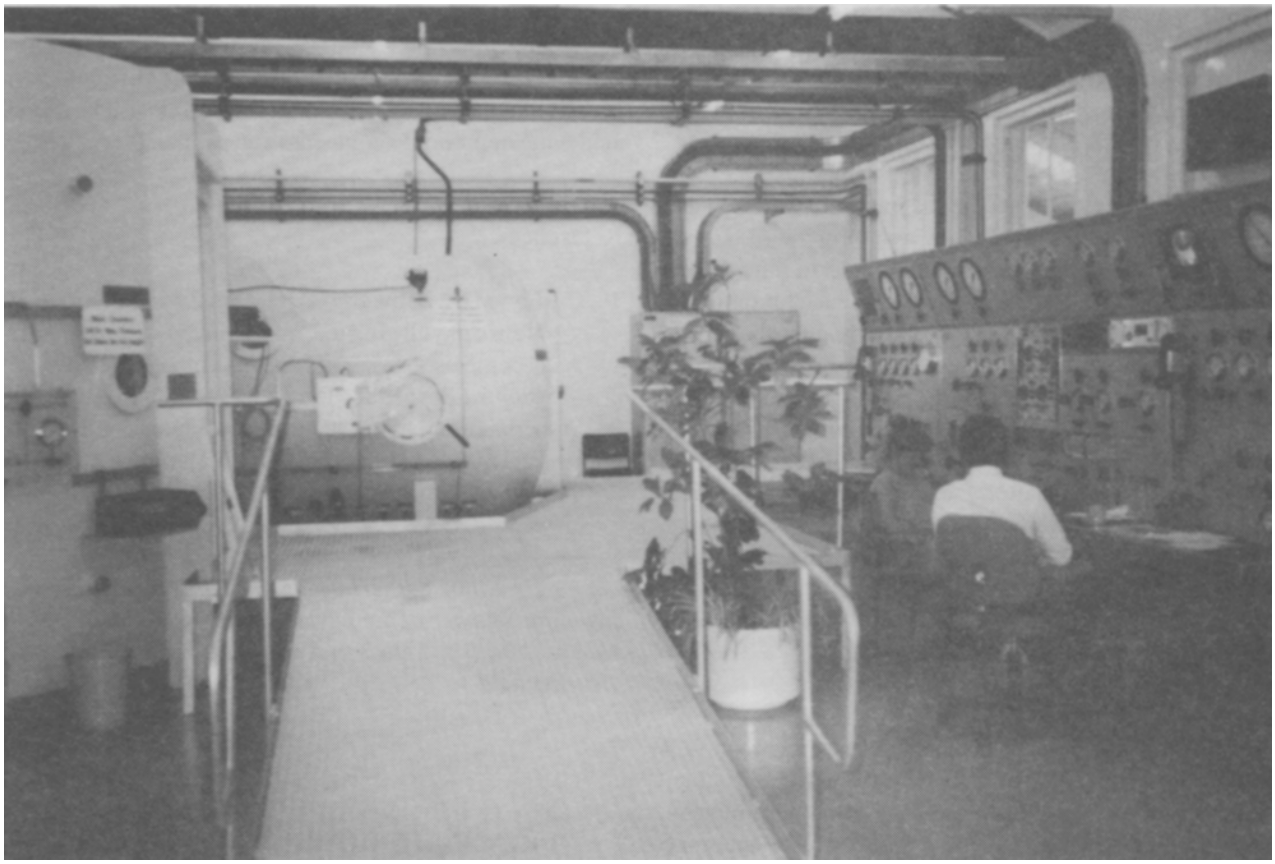

Figure 1. A general view of the Hyperbaric Facility. "Mildred" is shown in the left foreground with "George" behind. The control panel is on the right.

be carried in. The maximum operating pressure for this chamber is 3 ATA.

Normally patients are treated sitting in easy chairs with foot rests. Usually, four patients are treated morning and afternoon. Oxygen is usually administered by “Duke Hood” (Figure 2). Though Scott masks are available, they are more often used for treating divers. In an emergency, up to eight patients can be accommodated in this chamber. For comatose or intensive care patients, the chamber can take two trolleys with room for the attendants to move around it.

“George”, the other chamber, has an operating depth of 7 ATA and has been tested to 13.4 ATA. It is a horizontal cylinder 2 m in diameter with two compartments. The treatment end has a walk in door and a fold down bunk on each side. With patients sitting on the bunks, a maximum of three plus attendant can be accommodated.

Access to both chambers can be made through aseparate entry lock between them. This can be rapidly pressurised if required. A transportable chamber can be locked on to this entrance as it is fitted with a standard NATO mating flange.

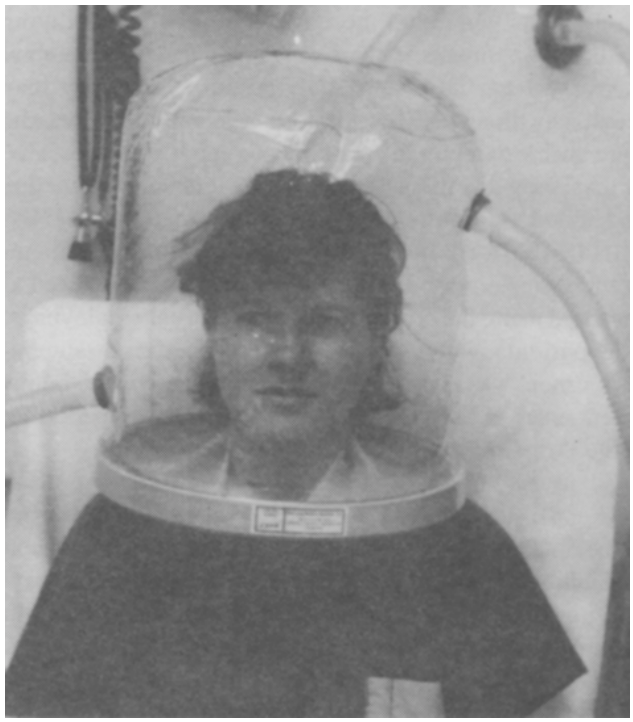

Figure 2. The Duke hood on a patient.

Monitoring facilities are available in both chambers. This permits continuous monitoring of any modality in any patient transferred from the Intensive Care Unit. There are 72 channels available in each chamber.

Case Load

187 cases were treated from 31.11.89 to 31.12.90. There were 1,808 patient treatments in 806 chamber runs.

82 emergency cases have been treated to June 30th 1990. These have included divers, crush injuries, carbon monoxide poisonings, surgical flaps with poor circulation and head injuries. For these the average number of patient treatment was 3.7.

Non-emergency cases were made up of non-healing wounds, radiation necrosis and assorted ulcers. This group need many more treatments per patient, the average has been 29 and one patient required 71 treatments.

In Western Australia, the diving casualties fell into three main groups; pearl divers from Broome, professional divers and diving fishermen, and recreational divers and their instructors. 41 divers have now been treated since the chamber was opened.

Research

There are two research projects currently in progress. The first is to investigate the role of complement as a predictor of susceptibility to decompression sickness.

The second project is to study the effects of hyperbaric oxygen on cardiac ventricular wall movement in acute myocardial disease, such as infarction or cardiomyopathy. Other research projects planned include a study of stroke patients.

Staff

The Unit has a staff of Director, three nurses and two technicians (one part-time) who have had wide experience in commercial diving. Five other physicians with training in Diving and Hyperbaric Medicine, including the Directors of the Intensive Care Unit and the Emergency Department, assist in providing round the clock cover. The registrar medical staff of the Emergency Department also participate in the care of our patients. A pool of about 20 nurses trained in hyperbaric nursing, drawn from critical care areas of the hospital, provides extra staff for in-chamber patient care.

A close working relationship has developed with the Director of the Emergency Department, and all of his staff, which provides early referral of acute cases, as well as, extra cover for the chamber operation.

Department case discussions are conducted so that all members are aware of what patients are being treated and their progress.

Discussion

Many years of research and discussion have resulted in a range of accepted indications for hyperbaric medicine being published by the Undersea and Hyperbaric Medical Society. As a result cases do not all have to be in double blind trials, as the effects of treatment have already been proven elsewhere.

As review of our clinical results is to be presented at the 1991 Annual Scientific Meeting by Dr Davies these will not be discussed in this paper. This paper will appear in a later issue of the SPUMS Journal.

Patients suffering from carbon monoxide poisoning who have a neurological deficit on presentation usually receive three treatments, often with rapid improvement in their mental state and resolution of other symptoms.

The close relations the Unit has with the Emergency Department in the hospital has ensured that acute crush injuries are transferred rapidly to the chamber.

One patient arrived from a remote area, 24 hours after a crush injury to the forearm. There was marked swelling, anaesthesia below the wrist with lack of movement of the fingers. Power and sensation returned during the first treatment and after three days of twice daily recompression he was discharged home symptom free, having avoided surgery completely.

Two other patients with crushed feet received great benefit, having previously been advised that amputation was probable, swelling was rapidly reduced and oxygenation increased.

Skin flap surgery, in which the vascularity of the flaps is compromised, can benefit from hyperbaric oxygen. This serves to reduce oedema and tissue tension, improve perfusion and may obviate the need for surgical revision. Short term treatment, twice daily, is usually effective for these patients.

Selected non-healing wounds benefit from multiple treatments but may require several weeks of therapy, six days a week. Often these patients have been unresponsive to prolonged normal therapeutic measures and and are referred as a last resort. Peripheral ulcers are variable in their response but do best if there is a good, large vessel blood supply to the area.

Vasculitis as a result of radiotherapy delays healing and healing can be assisted by hyperbaric oxygen. Radiation osteonecrosis, especially of the mandible, has responded well to this form of treatment, after which effective surgical reconstruction is practical.

Our experience with pearl divers is that the incidence of decompression sickness is relatively low considering the repetitive nature of their diving and the long times they spend each day at depth. Dysbaric illness is usually associated with a lapse from their normally accepted diving practice.

Other developments in Western Australia

Other "professional" divers and diving fishermen perform frequent repetitive dives, often paying only lip service to accepted tables. Many give a history of "getting away with it" for some years. Detailed enquiry often reveals stories of paraesthesia, niggles and extreme fatigue especially early in the season.

In the recreational group of divers, the experienced divers have been those undergoing advance courses such as for dive Master qualification. They usually dived, technically within the accepted tables, but had exacerbating factors such as hard work, fatigue, hypothermia or strenuous activity after the dive. Student divers were more likely to be fatigued with dives near the limit of the tables.

Notable among the divers was the small number of pure type 1 decompression sickness. Careful examination usually elicited paraesthesia, numbness, problems with balance, alterations of concentration and short term memory loss. A number of patients reported that during recompression they felt as if a veil had been lifted. They were unaware of how much their concentrations and thought processes were impaired.

The Pearl Producers Association is installing a chamber at the Broome District Hospital and a training course for the staff has been conducted by our Unit. We will be able to provide further support when the chamber is commissioned. The Broome chamber will provide a significant improvement in diver support in the North West of the State and will reduce the need to transport injured divers over 1,200 km to chamber facilities in Perth.

Conclusion

The Hyperbaric Medicine Unit at the Fremantle Hospital is providing a valuable service to the West Australian community. Apart from treating divers and many other medical and surgical conditions the Unit has conducted courses for hyperbaric nurses, doctors, Commercial Diving Supervisors, chamber attendants and for a group of pearl divers learning to operate their own chamber. There are frequent visits by diving clubs and other interested groups. SPUMS members visiting Western Australia are invited to contact the Unit and inspect the facilities.

Dr Harry Oxer is Director of the Hyperbaric Medicine Unit at Fremantle Hospital, Alma Street, Fremantle, Western Australia, 6160.

Dr David Davies is a visiting specialist on the staff of the Hyperbaric Unit. His address is Suite 6 Killowen House, St Anne's Hospital, Ellesmere Road, Mt Lawley, Western Australia, 6050.

Dr David Davies presented a paper on the clinical cases treated in 1990 at the Fremantle Hospital Hyperbaric Unit at the 1991 Annual Scientific Meeting. This will appear in a later issue.

THE DIVING MEDICAL AND REASONS FOR FAILURE

John Parker

Introduction

This is one medical practitioner's review of two hundred consecutive sports diving medical failures in an attempt to detail why divers fail the medical assessment.

The divers

The majority of the divers were young international tourists travelling through North Queensland to dive on and experience the Great Barrier Reef. The average age was twenty five (Table 1). Twelve hundred (59%) of the divers were male and 851 (41%) were female.

TABLE 1

AGE DISTRIBUTION

<20 years	9%
20-29	78%
30-39	10%
40+	3%

The medical

Every diver filled in a full questionnaire, as described

in Australian Standard 2299 diving medical form,[1] and had a full diving examination.

Spirometry involving the measurement of the forced vital capacity (FVC) and the forced expiratory volume in one second (FEV_1) was performed. Any diver with abnormal respiratory function was given 5 mg nebulised salbutamol and retested. The urine was tested for proteinuria and glycosuria.

A chest X-ray was taken if there was a personal history of lung disease, serious lung infection or recurrent lung infection, a family history of tuberculosis, a suspicious occupational history, a history of mechanical ventilation of the lungs, and any abnormality found on clinical examination of the respiratory system or poor lung function test results.

A resting electrocardiogram was performed on all divers aged 45 years or over.

An air conduction audiogram was performed on all divers with a history of reduced hearing, clinical evidence of poor hearing or a clinically abnormal tympanic membrane.

All female divers were asked if they were late for their menstrual period and if pregnancy was possible. If so a pregnancy test was performed.

The results of the first 1,000 medicals have previously been published.[2]

Results

The 200 diving medical failures were extracted from 2,051 consecutive initial sports diving medical examinations giving an overall failure rate of 10% (Table 2). One hundred and forty three (71%) of the failures were classed as permanent failures, i.e. the person should never scuba dive, and 57 (29%) were classed as provisional, i.e. the reason failure was potentially remediable, or further investigation might reveal the reason of failure to be invalid. Of the 200 divers who failed 181 (91%) had only one reason for failure, 17 (8%) had two reasons for failure and 2 (1%) had three reasons for failure. A total of 221 reasons for failure were identified.

Discussion

The reasons for diving medical failures are numerous. Forty seven different reasons for failing a sports diving medical were found in this series but this cannot be claimed to be exhaustive. There is still need for the compilation of a handbook to guide doctors undertaking diving medicals as specific indications and cut off points for failure or need of

TABLE 2

REASONS FOR FAILURE

RESPIRATORY		
Asthma	88	
Lower respiratory tract infection	12	*
Poor lung function (no obvious reason)	11	*
Pneumothorax traumatic	6	
spontaneous	3	
Previous chest surgery	3	
Pleural adhesions	2	
Pulmonary cyst	1	
Sarcoidosis	1	
Congenital aplasia of the lung	1	
ENT.		
Non-patent eustachian tubes	13	*
Severe scarring of tympanic membrane	12	
Upper respiratory tract infection	9	*
Deafness	7	
Impacted wax in external ear canal	6	*
Otitis media	3	*
Middle ear effusion	3	*
Abnormal sinuses	3	
Active heyfever	3	*
Infected wisdom tooth	1	*
Perforated tympanic membrane	1	*
Sinusitis	1	*
Sapedectomy	1	
Tinnitus	1	
CARDIOVASCULAR		
Arrythmias	3	*
Hypertension	2	*
Anaemia	1	*
Aortic stenosis	1	
Recent myocardial infarct	1	
Ventricular septal defect	1	
NEUROLOGICAL		
Epilepsy	2	
Previous intracranial surgery	1	
History of spinal cord injury	1	
Migraine	1	
DRUGS		
Bleomycin	1	
Warfarin	1	*
OTHERS		
Diabetes	2	
Severe caries	2	*
Severe herpes simplex infection of face	1	*
Pregnancy	1	*
Chronic active hepatitis	1	
Anxiety state	1	*
Severe scoliosis limiting respiratory function	1	
Inguinal hernia	1	*
Cold urticaria	1	
Obesity	1	*

* = provisional

further investigation or opinion. Indications for diving fitness have too long been left to the physician's personal prejudice, resulting in a wide variation of views, which therefore gave little credibility in the diving industry.

Conclusion

In 2,051 diving medicals 221 reasons for failing diving medicals were recorded in 200 potential divers giving an overall failure rate of 10%. Forty seven different reasons for failing the diving medical have been listed. Nine percent of divers have more than one reason for failing. A handbook of specific indications and cut off points for failure, further investigation or referral should be compiled.

References

1 *Australian Standard AS 2299 - 1979 Underwater Air Breathing Operations.* North Sydney: Standards Association of Australia, 1979

2 Parker J. Review of 1,000 sports diving medicals. *SPUMS J* 1990: 20(2): 84-87

Acknowledgements

The author sincerely wishes to thank Mrs Janette Downes for assistance in collating the data.

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THE ASSESSMENT OF THE PADI RESORT COURSE QUESTIONNAIRE

John Parker

Introduction

It is common practice for people undertaking scuba introductory or resort courses to go scuba diving without a diving medical, only completing a questionnaire on their health. The commonest diving instructor body undertaking resort courses in north Queensland is the Professional Association Diving Instructors (PADI). In the 1990 "PADI DISCOVER SCUBA AND INTRODUCTORY COURSE STATEMENT" sixteen questions are asked, preceded by the statement in bold letters "**If any (1-16) of these items do apply we regret that you must consult a physician prior to partaking in a scuba experience**". I used the information given in the Australian Standard AS2299 questionnaire[1], from 2,051 consecutive initial sports diving medicals, to complete an introductory course questionnaire for each diver to assess the efficacy of the resort course questionnaire.

Method

From the information given in their AS2299 diving medical questionnaire (and only that information) I completed the PADI course statement for all 2,051 divers. The questions appear in Table 1.

Results

Of the 2,051 divers 673 (33%) failed the resort course questionnaire and should have consulted a physician. Of these 673 divers only 142 (7%) actually failed the full diving medical I performed. Ironically only 106 (5%) failed the medical for the actual reason shown on the PADI questionnaire, the rest having problems not identified by the questionnaire. Hence the questionnaire only predicted 53% on the actual failures. See Table 2.

Discussion

The resort course questionnaire predicted 53% of failures compared to the AS2299 diving medical questionnaire which, when combined with an interview, predicted 63% of failures.[2] Questionnaires alone are therefore not a good predictor of diving fitness.

Discussion with local diving operators indicate that very few divers actually fail the resort course questionnaire. Certainly in 10 years of diving medical practice I have not had many divers referred for failing their resort course questionnaire. This may indicate that:

1 The diver, being warned that ticking a box in the questionnaire requires them to have a full diving medical with its additional cost and inconvenience, is encouraged to answer falsely.

2 The requirement of only having to indicate in the affirmative encourages people to miss things out. Having to state either a "yes" or "no" is more discriminating.

3 People are more honest in questionnaires when they know it will be followed by an interview.

4 Diving instructors make medical judgements on the questionnaire and override the statement that any positive response must eventuate in a physician's consultation.

5 I exaggerated their medical conditions in my comple-

TABLE 1

PADI QUESTIONS

1	I am currently suffering from cold or congestion
2	I am currently taking medication
3	I have a history of respiratory problems or disease
4	I am diabetic
5	I have a history of heart condition
7	I currently have an ear infection
8	I have recently had an illness or operation
9	I have a history of a sinus problem
10	I am pregnant
11	I have asthma, emphysema or tuberculosis
12	I am claustrophic
13	I smoke a pack of cigarettes or more a day
14	I have had difficulty with my ears
15	I have a nervous system disorder
16	I am under the care of a physician or have a chronic illness

tion on the questionnaire.

The PADI questionnaire can certainly be improved but still many people would be scuba diving who are not considered fit.

The commonest diving injury to such divers is aural barotrauma from pre-existing upper respiratory tract and aural problems. But the presumed greatest danger will be from pulmonary barotrauma in persons with lung infections, undiagnosed asthma or other lung diseases.

This danger can be reduced by taking the resort diver underwater in calm still water, under the strict supervision of a diving instructor. However, the dangers are increased because of the absence of any substantial training resulting in a higher risk of panic and uncontrolled ascents.

To require all divers in a resort course to have a full diving medical would decimate the diving tourist industry in North Queensland and prevent a large number of tourists from having first hand experience of the Great Barrier Reef and the joys of scuba diving.

To stop the present practice of only having a health questionnaire would necessitate evidence, not only of the potential dangers but of actual cases of injury or death. At present there are no such reliable data on the morbidity or mortality of the resort course. In the meantime the questionnaire needs to be revised.

Conclusion

The PADI questionnaire is a poor discriminator of scuba diving fitness, identifying only 53% of divers who should have failed.

References

1 *Australian Standard AS 2299 - 1979 Underwater Air Breathing Operations.* North Sydney: Standards Association of Australia, 1979

2 Parker J. The Diving medical and reasons for failure. *SPUMS J* 1991; 21 (2):80-82

Dr John Parker's address is P.O. Box 207, Airlie Beach, Queensland.

TABLE 2

COMPARISIONS BETWEEN PADI QUESTIONNAIRE AND THE FULL DIVING MEDICAL IN 2051 DIVERS

	Number	% of "Failures"	% of Total
Failed the questionnaire	**673**		**33**
Failed the questionnaire and failed the full medical for the right reason	106	16	(5)
Failed the questionnaire and failed the full medical but for the wrong reason	36	5	(2)
False failures			
Failed the questionnaire but passed the full medical	531	79	(26)
Passed the questionnaire	**1,378**		**67**
Passed the questionnaire and passed the full medical	1,284		(62.5)
False passes			
Passed the questionnaire and failed the full medical	94		(4.5)
Totals	**2,051**	**100**	**100**

PERSONAL PAPER

A "BEND" BY ANY OTHER NAME

Anon

I was born in 1937. I am a graduate, Chartered Mechanical Engineer and ex-R.N. Commander. My diving experiences, which I am about to recount, started in the Far East when the ship's diving officer convinced me that there was much beauty to be seen under water and, with my agreement, ensured that I was taught to view this inner world in comparative safety ! I was hooked ! Every available hour for the remainder of my Naval career I was to be found submerged in some part of the world or other

When I left the Navy I decided to combine both my skills as an engineer and my hobby as a diver. I was employed by an international company as an underwater engineer whose function was to design, and fit, sub-sea equipment required by various oil companies.

To keep to the subject matter of this story, the first bend I had was while working in the North Sea at approximately 34 m. The job was to fit anti nets around the legs of an unoccupied platform about 40 miles off Yarmouth. We were diving from an old fishing trawler in untethered scuba gear. If my memory serves me right, we were on bottom for about 18 minutes with a 5 min stop at 9 m. We had been working on this exercise for two or three days. On the last occasion, I was working on these nets, when my demand valve fell apart in my mouth. The retaining ring around the second stage just snapped and I was left with a stainless steel funnel in my mouth. Needless to say, I did not hang around looking for all the component parts. I bailed out rather quickly. One had to be rather careful about coming up, because as the visibility was very poor, it was possible that one would come up inside the rig structure, with all its cross bracings etc. and this could become rather dangerous. So after getting my bearings, I swam out from under the rig and finned for the surface. Believe me it didn't take long !

When I came to the surface, I just climbed on board the trawler cursing and swearing about the equipment, undressed and had a shower.

There was no thought at all about decompression sickness, emboli and such, and anyway, we did not even carry a recompression chamber on board.

Whilst I was having the shower, I found that I kept being thrown against the side of the stall each time the ship rolled. Apparently after all my years at sea, I could not compensate for the movement of the ship. I found this a little disconcerting, so after getting dressed I found the Captain and requested him to start for shore. After about 30 minutes, I found that both my right arm and leg were becoming difficult to move, a classic hemiplegia. By now I was becoming perturbed at my increasing debility, so the Captain requested assistance from the Air Sea Rescue. I was picked up by a Wessex helicopter by strop from the deck, flown ashore and put into a pre-prepared recompression chamber. I was immediately blown down to 36 m, at which depth, the symptoms started to ease and over the next 48 hours they disappeared completely. I estimate that approximately two and a half to three hours from the time of the underwater incident to the time I reached 36 m in the chamber had elapsed.

I made a full recovery from this accident and after one month's compulsory leave, I recommenced diving.

In 1974, 18 months after the first incident, I was in West Africa working on a problem that an oil company was having with a submerged pipeline manifold system. This manifold was under approximately 21 m of water in visibility of around 15 cm. Just to enlarge upon the story, in case it adds any relevance to the eventual outcome, I had only been in West Africa for about two weeks. Before this job my last underwater exercise had been at 135 m off South Africa, working from a diving bell, using 90% helium and 10% oxygen. This had lasted for 3-4 weeks. I put this in because it may have contributed to a laissez faire attitude on my part, having come from that depth to a piddly 21 m. I don't know however.

We had just about completed work on the manifold. The new components had been fitted and we were about to start fitting the hoses, when the tanker, that we had been told was still 24 hours away, hove into view.

Now the conventional berth was being operated by Southern Europeans, lovely people, but not renowned for their calm acceptance of alterations to an established procedure.

In an attempt to get the operation on stream quickly, we started to do repetitive diving to get the hoses hooked up and tested. During this work, the tanker moored up in the berth overhead and our work boat tied up alongside her. Once everything had been connected, we pressurized the system to check for leaks and I went down to do a final survey. Everything appeared to be oil tight. This inspection probably took 5-10 minutes bearing in mind we were free diving and the visibility was still around 15 cm.

On my way to the surface I came up under the work boat (the hull had a draft of 2.4 m), so I finned out from under it and climbed up over the stern. While giving my report to

the berthing master I started to feel dizzy and the whole world began to spin. After that, I remember nothing. Until I regained consciousness, my responses and actions reported here are from hearsay only.

I was told that, as I hit the deck of the work boat, I started an epileptic seizure. When it subsided they put me in a sling and brought me up onto the deck of the accommodation vessel. I was still unconscious. They then put me on oxygen while I was still lying on the deck. There was a recompression chamber on board, but it was a "one man pot" 0.9 m in diameter and 1.8 m long, with only a tiny medical lock for passing in drinks etc.

It was decided by those who were in charge of my treatment, two merchant navy captains and three divers, that it would be unwise to put me in the chamber because if I started fitting again under pressure I could die, as no one could get into me unless they vented the chamber.

In the event I slowly regained consciousness lying on the deck. Unfortunately, the only thing I could move was my tongue. I appeared to be totally paralysed from the neck down. I assume my brain had had such a shaking from the fit that it could not focus on my immediate plight because it appeared to those about me that I accepted my condition with equanimity.

I remained on deck for 3 to 4 hours during which time my paralysis appeared to retreat progressively down my body. Firstly I could move my arms and then my trunk. It looked as if what ever trauma had occurred was dissipating and everything would be fine. So they would fly me ashore the next day. I was carried to bed, where I stayed for the next 4 to 5 hours. Then I tried to get up to use the toilet, I fell flat on my face. My legs just refused to work. They decided to get the helicopter out urgently and fly me ashore to the medical facilities there. At the same time they decided that perhaps I did need recompressing after all, so a request went out to another diving company (non-English speakers) for the use of their chamber. During the night they brought it to the medical centre with all its accoutrements, including all their divers, and into it I went. Again it was basically a one man chamber with no air lock, only a tiny 20 cm medical lock for drinks, urine bottles etc. There was still concern that I might start having seizures again so I was put in with a French speaking diver who had had some form of medical training. Sounds comfy ! This poor chap had to sit up tight against the door half on and half off my feet. But that did not matter because I could not feel them anyway! We were pressurized up to 36 m and there we stayed. It was now about 10 to 12 hours after I first lost consciousness.

By now the lumbering oil field was starting to roll forward. A work boat was assigned to take me from the medical centre at the river estuary to Warri (where there was a full hospital). The trip would take approximately 12 hours. The chamber and equipment was loaded on the vessel and away we went.

During this time, a large 6 man chamber was being prepared and fittings made, so that it would be already pressurized and the air lock open, ready for the time I arrive and be placed outside the front door of the hospital.

I was becoming more and more depressed about the whole affair. After having thought that the trauma was over when my recovery started, here I was with a Frenchman sitting on my legs, who could not speak a word of English. My bladder distended half way to my neck, and the situation was getting worse by the hour. You will note I did not give a thought to the poor chap who was sitting in a cramped position at one end of the minuscule decompression chamber with an Englishman who could not or would not speak to him and who just kept moaning and complaining in a foreign language !

Anyhow, when the work boat docked at the Shell camp in Warri, the complete chamber was lifted onto the back of a truck and transported to the hospital, where it was unloaded at the front door. I do not think you could see the front of the hospital for chambers and their equipment.

By this time my ascent to the surface was just about completed so the door was opened and I was put on a stretcher, rushed into the hospital for a neurological examination and, sweet relief, catheterisation. This examination only lasted a matter of minutes. Then without further ado I was carried out and placed in the large chamber for further treatment.

My psychological outlook had changed a little for the better at this point, as I thought at last somebody was aware of what was wrong and was doing something about it. A bit selfish and unfair perhaps, considering all the effort that had been expended on my behalf, but one was only thinking of oneself.

Once in the larger chamber, I started on a new decompression regime created at HMS VERNON, and telexed to West Africa. Now, I want the reader to bear the following in mind, the incident happened 16 years ago and I did not see, nor was I consulted about, the tables used. Moreover I was not viewing anything very objectively. So the depth and times stated may vary slightly from what actually happened, but the following is a fair approximation of the following events.

The new chamber was pressurised to 36 m, at which depth I stayed for 12 hours. We then started to the surface very gradually. When we reached 24-27 m I was started on oxygen, the regime was 30 min on, 30 min off. I continued this for four days until we reached the surface. In all, I was in the chamber for four and a half days. But with the full

sized air lock, I was getting fed and watered. I could have all I wanted now that I was catheterised. I had plenty of visitors and this alone brightened me up.

Despite all this effort and cost, there was no further improvement in my condition and my legs and other appendages (I told you I felt better) refused to respond to stimuli. I was flown home to England and admitted to hospital, where I remained on the Orthopaedic Ward (someone had said I had a back injury !) for twelve weeks. During this period, I was subjected to regular physiotherapy and slowly progressed going from a walking frame to axillary crutches, and then to elbow crutches. I will not bore you with the midnight Chinese feasts and the times I got stuck on the third floor at three in the morning. It was easy to climb stairs but impossible to come down. Needless to say, I was determined to walk again.

The physical situation now is that I still have a peripheral neurasthenia from the waist down and slight spasticity in the legs, but that does not appear to curtail me from whatever I wish to do. I am a qualified pilot, I ski (snow and water) and ride to hounds. I am the proud and pestered father of two daughters (although the impotence took some working on) and am happily married, though if I was my wife I would have left me years ago ! My daughters incidentally, have just completed a diving course and they are as enraptured by the sport as I was. I have no qualms at all about them following the activity.

I hope this story will be a salutary lesson to all divers. Accidents happen at all depths, so be ever watchful and have the equipment available or responsible parties contactable at all times.

Happy diving.

SPUMS NOTICES

MINUTES OF SPUMS EXECUTIVE MEETING (TELECONFERENCE), FEBRUARY 3, 1991 AT 1000 EST

Apologies

Dr Grahame Barry

Present

Drs Des Gorman (President), John Robinson (Secretary), John Knight (Editor), Tony Slark (Past President), David Davies (Education Officer), Chris Acott, Darryl Wallner and John Williamson.

Absent

Dr Chapman-Smith

1 Minutes of the previous meeting

The minutes, already circulated, were taken as a true and accurate copy of the previous meeting. Proposed Dr Stark, seconded Dr Gorman.

2 Business arising from the Minutes

2.1 AGM 1991 MALDIVES

A letter from Dr Pease, suggesting that the costs for attending members at the AGM is higher than that on offer at other travel agents, had been circulated. The Secretary had already informed Allways Travel of the nature of the letter. A formal reply from Adrienne McKeon had been received by the Secretary outlining the reasons, which in fact had been presented at the previous AGM, why large groups cannot get the travel concessions available to small groups. It was decided to obtain Adrienne's permission to publish this letter in the forthcoming SPUMS Journal to lay this issue to rest.

Dr Gorman reported that the programme for the AGM had been finalised. A copy was going to be forwarded to each presenter, and to Dr Knight for publication in the next Journal, before the meeting.

A letter from Dr Chapman-Smith was read indicating reservations about the Maldives as a destination with because of the Gulf War. No other member of the Executive shared Dr Chapman-Smith's reservations. It was decided to continue with the AGM as planned. Dr Gorman reported that the programme had been put in place, and that the invited speaker, Dr Glen Egstrom, was very keen to come. A letter from Dr Knight, suggesting that oxygen be available on all dive boats, was circulated. He felt SPUMS should set an example. The Secretary had already communicated Dr Knight's concerns to Allways Travel and had had a letter from the dive boat operator who will make every effort to fulfill this requirement. The difficulties of providing oxygen in a third world country were noted. Dr Williamson pointed out that availability of oxygen was irrelevant as we did not know what capacity and what method of administration from such oxygen cylinders was available.

Dr Knight suggested that a Safety Sausage be

given to each diving participant. This was agreed to by the Executive and the Secretary was instructed to inform Allways Travel accordingly.

2.2 AGM 1992 PORT DOUGLAS

Dr Williamson reported the result of investigation of costs at the Sheraton Mirage Hotel compared to the Radison Royal Palms Resort. The costs of the Mirage were such as to effectively exclude it from consideration as the venue for this conference. However, if members wished to be at the Mirage there is no reason why they should not avail themselves of that facility on a private basis. It was agreed by the Executive that the invited speakers should be accommodated free for two nights at the conference venue.

Further discussion ensued as to the format of the meeting. It was agreed that it should be arranged around the proposition of four days of full conference and three days of diving from Quicksilver with a mini-programme on these diving days. The suggested format was perhaps a half-hour lecture before dinner. Presentations on the boat were ruled out because the facilities were not adequate. Dr Williamson said that Michel Pichon has a programme well in hand. Dr Williamson will proceed with more detailed planning of the programme plus dates and report to our next conference. Dr.Williamson stated that if any topic was thought desirable to add to the programme he is to be notified. The target date for the conference was assumed to be June 1st.

2.3 HYPERBARIC SOCIETY

Dr Williamson reported that the group is now known as the Australian and New Zealand Hyperbaric Medical Group (ANZHMG). The first meeting has been held and agreed that the organisation should come under the umbrella of SPUMS. Appreciation was expressed by the group for SPUMS assistance and in particular for financial assistance by paying for the teleconference. The next meeting will be on Wednesday March 20th at 1000 EST. The ANZHMG Secretary is Dr David Tuxen and the Chairman is Dr John Williamson. Dr Williamson reported that a great deal of data had been circulated by Dr Tuxen and he was very satisfied with the progress, however Dr Tuxen will be in the United States in the next six months. He will continue his role in the ANZHMG at his own expense. It was suggested that the ANZHMG could hold its first AGM at the Port Douglas meeting of SPUMS.

Dr Davies suggested that the SPUMS Education Officer should be involved in the ANZHMG in the future, particularly as, in relation to the awarding of the Diploma, he would need to know who has done what and in which units. In addition it frequently falls to the Education Officer to notify what jobs are available. Dr Williamson agreed that the Education Officer should be involved. Dr Gorman indicated that proceedings of the group would be published in the SPUMS Journal.

2.4 COFFS HARBOUR MEETING

Dr Wallner reported that an attractive programme had been arranged and that he had about 20 participants. Some discussion ensured as to the reporting of the conference in the Journal. It was felt at this stage that all presenters should submit for publication although publication cannot be guaranteed. There is now sufficient material for publication that the Editor will only include material in the Journal after peer review. However, Dr Gorman pointed out that invited speakers at the AGM would automatically receive publication.

2.5 THE STANDARDS AUSTRALIAN C.S.83 COMMITTEE MEETING SYDNEY, 13TH DECEMBER 1990

Letters from John Knight, Bob Thomas, Colin Melrose, Des Gorman and John Williamson have been circulated to the members of the Executive. A number of changes had been made to the draft standard and these had been circulated. The Committee accepted the draft standard as altered and also accepted the notion of a two-year window to allow people who are currently doing diving medicals to obtain satisfactory qualifications to meet the new standard. In general the changes were acceptable. However, the Committee strongly expressed the view that SPUMS must maintain its firm stand that people doing medicals must be of an acceptable standard of training and that there be no backsliding on this matter.

The SPUMS censors are the President, the Director of an active Hyperbaric Unit and one other person. At this moment these are Dr Des Gorman (President), Dr John Williamson (Director of an Hyperbaric Unit) and Dr David Davies (Education Officer). After much discussion it was agreed that the prime role of the censors should be awarding the Diploma. It is open to any body, government or otherwise, to use the Diploma as an indication of training to a necessary standard. However the Society has already accepted that the censors are to be involved in approving the content of diving medical courses for purposes of the proposed standard and will carry out this obligation until other regulatory bodies are appointed.

2.6 REORGANIZATION OF SPUMS SECRETARIAT

The Secretary reported that the Science Centre continued to charge large sums of money including a

December 1990 account of $247.00. Drs Barry and Robinson had discussed this matter and neither were able to ascertain what work, if any, the Science Centre had done to justify this account. However a decision was made to pay on this occasion.

The new permanent address of SPUMS c/- The Australian College of Occupational Medicine, P.O. Box 2090, St Kilda West, Victoria 3182, Australia.

In future all mail will be addressed to the College of Occupational Medicine and on a weekly basis will be bundled up and forwarded to the office centre of the Secretary's choice. Dr Robinson has chosen the Brighton Executive Centre. The centre has undertaken to process mail. They will forward application forms to people who request them along with the standard accompanying letters. Completed application forms, with the correct sum of money, will be forwarded to Dr Barry. All other correspondence will be forwarded to the Secretary for his attention. After Dr. Barry processes an application it will then be forwarded to Dr Knight for entry in the list of members. Records of members will be kept by Dr Knight, the Editor of the Journal, on his computer. Processed application forms will be sent the Secretary for filing. This system is able to continue wherever the Secretary resides as it is merely a matter of informing any change of address to the Australian College of Occupational Medicine. It gives SPUMS the great benefit of having a permanent and fixed address.

Dr Gorman indicated he was delighted with this new address. It has obvious implications of leading on to what other role the Australian College of Occupational Medicine may play in the Society. In particular, they may wish to co-sponsor our Diploma.

3 Other Business

3.1 INDONESIAN MEETING

Dr Robinson raised the suggestion that members attending overseas meetings should give a short address at the Annual Scientific Meeting and these should be published in the Journal to keep members, who are unable to go to the ASM, up to date with what is going on around the world. The Executive considered this was a good idea and Dr Gorman indicated he would be amending the ASM programme at the Maldives to cater for this.

4 Correspondence

A letter was circulated from Divevac, South Africa. The Executive decided that the letter be published in the Journal but that no specific action need be taken.

5 Business without Notice

Dr John Williamson spoke about the ANZ Thoracic Society meeting in July at Lorne, Victoria. Professor David Dennison from London, who has different views on respiratory fitness for diving to those held by the Society, is to speak. Dr Andy Veal, a SPUMS member, will be presenting, very strongly, the views commonly accepted by diving physicians in Australasia.

Dr Knight suggested that SPUMS should donate to the Diver Emergency Service. He suggested that $5.00 from each Australian subscription should be paid as a subscription for Dive Safe magazine to be sent to members. Further discussion on this item was postponed to the next meeting.

There being no other business the meeting closed at 1145 EST

ALTERATION TO THE RULES OF THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY INCORPORATED

At the 1991 Annual General Meeting the following three motions were passed unanimously.

Motion 1

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

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

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

Motion 2

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

Rule 41 (b) would then read

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

Motion 3

"That rule 4 be altered by deleting paragraphs a to h, and substituting paragraphs a to f below."

Rule 4 reads

"(a) Any person seeking full membership or associate membership or corporate membership may apply for membership in the form set out in Appendix One which shall, after it is completed, be lodged with the Secretary of the Association.

(b) As soon as it is practicable after the receipt of a nomination, the Secretary shall refer the nomination to the Committee.

(c) Upon a nomination being referred to the Committee, the Committee shall determine whether to approve or to reject the nomination.

(d) Upon a nomination being approved by the Committee, the Secretary shall, with as little delay as possible, notify the nominee in writing that such a person is approved for membership of the Association and request payment within the period of 28 days after receipt of the notification of the sum payable under these Rules as the entrance fee and for the first year's annual subscription.

(e) The Secretary shall, upon payment of the amounts referred to in sub-clause (d) within the period referred to in that sub-clause, enter the nominee's name in the register of members kept by him and, upon the name being so entered, the nominee becomes a member of the Association.

(f) A right, privilege or obligation of a person by reason of his membership of the Association -

(i) is not capable of being transferred or transmitted to another person; and
(ii) terminates upon the cessation of his membership whether by death or resignation or otherwise.

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

(h) The Committee shall be under no obligation to give any reason for its decision not to accept an application for membership of the Association."

The words to replace the paragraphs above are:-

"**(a) Any person seeking full membership or associate membership or corporate membership may apply by writing to SPUMS Membership, C/o Australian College of Occupational Medicine, P.O.Box 2090, St Kilda West, Victoria 3182.**

(b) When the completed application form, with the sum payable under these Rules as the entrance fee and for the first year's annual subscription, is received by the Treasurer the applicant's membership shall commence.

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

(d) A right, privilege or obligation of a person by reason of his membership of the Association -

(i) is not capable of being transferred or transmitted to another person; and
(ii) terminates upon the cessation of his membership whether by death or resignation or non-payment of subscription or otherwise.

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

(f) The Committee may reject any new member at its next meeting and shall be under no obligation to give any reason for its decision not to accept an application for membership of the Association."

The motions now have to be passed by a three fourths majority of the full members and life members in a postal ballot.

As on previous occasions when the constitution has been amended it is intended to hold a postal ballot only if the Secretary is notified in writing that any member wishes to oppose any one of the three motions.

If no objections are received by the Secretary before September 14th 1991 it will be assumed that the motions have been passed by the necessary three fourths majority of full and life members.

If an objection has been received ballot papers will be posted to all full and life members.

Darryl Wallner
Secretary SPUMS

REPORT ON COFFS HARBOUR MEETING

The meeting was marred by poor weather which restricted diving to Sunday only. However, the Aanuka Resort and its environs made up for this disappointment.

The lecture programme was well attended with 48 participants. Particularly welcome were local divers, instructors and medical practitioners, some of whom had come considerable distances.

Steve Smith, a marine biologist, gave a most informative talk on the uniqueness of the Solitary Isles.

Alan Bridger's "The middle meatus and sinus problems in diving" was an excellent presentation of a practical and new approach to this important subject.

Unfortunately Cathie Shannon was unable to attend personally, but she provided three case histories of decompression sickness which provoked lively discussion.

Carole Wright gave an interesting talk on "Problems with divers from the instructor's viewpoint". This paper reinforced our role as medical examiners in presenting candidates who are "fit to dive".

Peter Lewis presented a series of diving fatalities from New Zealand in a thought provoking manner which engendered lively debate.

Finally, Larry Thornton gave a superb slide presentation of "Diving in the South Pacific".

The general response to this meeting was most favourable and there appears to good support for further meetings of this type which can contribute to SPUMS educational role in a regional manner.

Darryl Wallner
Organiser

Dr.D.P. Wallner's address is 114 Vasey Crescent, Campbell, A.C.T. 2601, Australia.

LETTERS TO THE EDITOR

MARINE STINGER GUIDE

Queensland Surf Rescue
P.O. Box 36
Newstead Queensland 4006
12 April 1991

Sir,

Queensland Surf Life Saving is please to announce the publication of its new educational package on Marine Envenomation. Produced by Dr Peter Fenner, a world authority on marine envenomation and the Association's Marine Stinger Officer, the Guide, posters and video give simple, but most up-to-date information on the identification and treatment of stings and bites from many of our marine creatures.

The "Marine Stinger Guide" is a pocket-sized reference book produced at the very reasonable price of $Aust 8.00 (plus P&P) with excellent photographs of the creatures and their envenomations. Designed specifically for the first-aider or person involved with the sea in work or leisure, the text is kept brief but factual and has all the most recent information available. As well as descriptions and photographs of the creatures, it describes and illustrates the envenomations and gives effective first aid treatments. There are also relevant sections on awareness and prevention of envenomation.

Two large wall charts on jellyfish envenomation complement the Guide. One is on "Jellyfish Identification" and using the same illustrations as the Marine Stinger Guide, identifies the jellyfish and its sting pattern. The other deals specifically with the treatment of jellyfish stings. The cost is $Aust 3.00 each (plus P&P).

Later this year, the "Marine Stinger Book" will be available in its 4th edition. It will be much larger than the previous three editions and contain comprehensive details on a wide range of marine envenomation problems. Designed this time as a specific reference guide for the medical fraternity or people scientifically involved or interested in this area, the book will be edited by Drs John Williamson, Peter Fenner and Professor Joseph Burnett. It will also have a number of chapters written by specific experts in field.

Please contact us for further details, or forward an order form to us at above address.

Brett Williamson
Surf Lifesaving

A review of the MARINE STINGER GUIDE appears on page 91 of this issue.

DIABETIC DIVERS

Suite 12, 8 Pacific Parade
Dee Why, NSW 2099
20/3/91

Sir,

I am writing to seek help from your members in a research programme that I am developing into the number of divers who have insulin dependent diabetes, the number of dives they have done and the diving complications they suffer. I would also like to hear from those who have had no complications.

I know there are many such divers and I would like to hear from them, or from doctors who know of them, and what their experiences have been. If they could write to me and include their telephone number I will get back to them.

All information will be treated in total confidence.

D W Richards
MB, BS, FFARACS

BOOK REVIEWS

THE MARINE STINGER GUIDE
Surf Life Saving Association of Australia. 1991
Price $ 8.00. Postage and packing extra.

JELLYFISH IDENTIFICATION (Poster)
TREATMENT OF JELLYFISH STINGS (Poster)
Price $ 3.00 each. Postage and packing extra.

The Marine Stinger Guide lives up to the expectations raised by the letter on page 90. It is a short (57 pages), easily read small book (11 cm x 21 cm) which fits easily in a deep pocket or a beach bag. It deals with stinging and envenomating animals in a logical format. First a picture of the animal, then its distribution, common names, size and appearance, the symptoms and signs with clinical pictures and finally treatment. The pictures are excellent and beautifully reproduced in colour. Besides the pictures of patients there are pictures showing the recommended treatment for cardio-pulmonary resuscitation (CPR), the Surf Life Saving "Stinger Drill" which is 1 retrieve and restrain, 2 douse with vinegar, 3 assess consciousness and 4 apply compression bandages. There is a very clear series of drawings showing the way to apply compression bandages.

Besides the common dangerous jellyfish, with appropriate emphasis on *Chironex*, the book covers the blue ringed octopus, stonefish, stingrays and sea snakes with information about the injuries they can inflict and the appropriate treatment.

This book should be in the beach kit, or the diving bag, of anyone entering the seas of northern Australia.

The two posters are large, 1 by 0.7 m, and well suited to prominent positions in public buildings on northern beaches and the club house of any organisation that uses those seas. The Treatment of Jellyfish Stings poster includes one of the best instructions for CPR that I have seen.

John Knight

BOOK REVIEW

A PICTORIAL HISTORY OF DIVING.
Best Publishing Company, P.O. Box 30100, Flagstaff, Arizona 86004, U.S.A.
Price $US 83.00, postage $US 6.50.
Mastercard and Visa credit cards accepted.

This publication is the result of an UHMS initiative and is best summarised, in the words of the Preface, as an illustrated history of the evolution of diving equipment. Do not be dismayed by this because it has been the development of the equipment which has determined the development of diving from its original simple breath-hold mode to the present sophisticated range of options, from scuba to saturation systems, from habitats and submersibles with lock-out facilities to 1 ATA diving suits.

This is far more than a coffee-table book containing a collection of (excellent) coloured illustrations, because each chapter has a text which concisely describes the subject covered, each written by a different author. The authors are all respected and acknowledged leaders in the world of diving. Even more relevant is the fact that each of them appears to have been able to extract the essential facts of their subject and present them succinctly and in a readable manner. This is no mean achievement. It is a book which repays a close, and even repeated, reading to realise the amount of data contained in the text and the descriptions of the objects illustrated.

There is naturally only passing reference to the numberless brave men who used the old-time equipment but it is clear that their morbidity and mortality must have been at a level which we, nowadays, would never tolerate if it became known. It is easy for us to think of our presently accepted understanding of the physiology and pathology of diving-related problems as having been common knowledge from time immemorial, whereas in actuality it has been painfully acquired over comparatively recent years.

In reading this book think sometimes of the men who worked from open bells in cold, dark waters and their successors who depended on unreliable air pumps for their next breath, whose helmets might either fall off or fill with water if they stumbled. Our recreational (and commercial) diving is an elegant (and safe) descendant of such rude ancestry and represents the fruit of the interaction between innovative engineers (some of whom dived in their own creations) and the humble people whose bodies served as the test-beds for their bright ideas. Never forget that this process continues to the present day, though modern divers are no longer so uncomplaining nor so accepting of risks and problems. One has only to consider the constant production of "improved and safer" dive tables to understand that the process, of divers acting as the subjects for the trials of new ideas, continues. This procedure has been made much safer by the practice of seeking feed-back of information to engineers and inventors from the users (divers) and the medical person involved in the management of the results of erroneous practices. It is on this triad of interests that improvements in diving safety ultimately depend.

Naturally there is a drawback to this book. It is the cost. And the Editor might have exercised a tighter control over the enthusiasm of some of his authors for their subjects. One can even have too many pictures of those marvelous copper and brass helmets. This is a book to grace any library. It is intended as a compliment to the authors to say that the reader may be left with a desire to learn more about the men and equipment whose troubled history has brought us to our present informed position on the major problems of present day diving.

Douglas Walker

SPUMS ANNUAL SCIENTIFIC MEETING 1990

PSYCHIATRIC ASPECTS OF DECOMPRESSION SICKNESS

Chris Acott

Introduction

Over the past 10 years our understanding of and the treatment regimes for decompression sickness (DCS) have been gradually changing. Perhaps perfluorocarbons, SPEC scanning and the use of helium will be common place in the therapy of decompression sickness (DCS) in the 1990's.

There is increasing evidence that DCS primarily involves the central nervous system (CNS) and that pure Type 1 lesions are rare. Figures for involvement of the CNS range from 10 - 30% in the 1970's to 78% in a series from the school of Underwater Medicine at HMAS Penguin in 1985-86. Indeed, in another series up to 50% of Type 1 DCS were shown to have abnormal EEG's which took up to a month to normalize.

Recently, an article in the Lancet[1] showed that all Type II DCS cases had cerebral perfusion deficits shown by SPEC scanning. Therefore, it may appear that the diagnosed involvement of the CNS is dependent upon the thoroughness of the CNS examination. If one does not look for it one does not see it.

Other recent publications allude to the "punch drunk" diver, and the late neuropsychological changes occurring with diving and DCS. This has received a lot of attention in the literature. However, at present I feel that all these can be summed up by Hayward in "The Long Term Neuropsychological Effects of Compressed Air Diving". "The issue of whether DCS or various indices of dive stress are associated with decrements in neuro-psychological function still remains unresolved. It seems likely if there are effects on neuro-psychological functioning due to exposure over time to risks taken when diving they are probably not large (at least in the short term) and probably only affect a subset of individuals"

There is no data available on the acute psychiatric, or psychological manifestations of DCS, except perhaps, the mention of personality changes noted with cerebral DCS. There are numerous anecdotal reports, particularly those associated with the abalone divers, that acute psychiatric changes occur. I have talked to some of the shellers who remain in the boat while the diver is below. Their reports indicate that acute personality changes occur and these vary with the particular dive profile. The shellers could differentiate the depth of the dive on the mood and personality change seen in the abalone diver upon surfacing. These stories were so constant that one would have to assume that they are true. Acute confusional states were seen with deeper dives while aggressive, abusive behaviour was seen with the longer shallower dive.

There is often considerable delay between the onset of the symptoms of DCS and the actual time the diver presents himself for treatment. This delay is frequently attributed to:

1 Ignorance of the meaning of the symptoms both in the diver and the dive leader.
2 Over indulgence in alcohol.
3 The symptomatology being blamed on a previous injury.
4 Denial. The dive was well within the limits of the tables so the diver could not possibly be bent.
5 Guilt. DCS is regarded in some circles as shameful as an STD.

Failure to recognize that there is something wrong may, in fact, be a manifestation of the disease. Unrealistic or perhaps, in some instances, a paranoid reaction to the symptoms may in part be part of the disease itself. This is sort of a "Catch 22" situation. To recognize that one has DCS one must recognize the symptoms, but one of the symptoms of DCS is that one does not recognize that one has got it.

This brings me to my topic, which, could be called The Post DCS Blues and DCS Psychosis.

Since joining the Hyperbaric Unit at the Royal Adelaide Hospital (RAH) I have made the clinical observation that most post treatment patients go through a depressive phase, and that divers diagnosed as having cerebral DCS have a degree of psychosis, manifesting itself mainly by paranoid behaviour. The following case histories will demonstrate this.

Case Histories

Case 1

This 32 year old diving instructor had been working in the Maldives. He presented at our department on the 27th of September 1989. He had been flown down from Darwin Hospital by the Royal Flying Doctor Service in a pressurized aircraft.

He was agitated, irritable, slow and vague. He had poor short term memory. He was extremely unstable on his feet with a sharpened Romberg of 2 seconds. When doing serial 7's he made 4 mistakes in 120 seconds, while serial 5's took 60 seconds. He had worked in a statistics department before going to the Maldives).

CT scan, EEG and chest X-ray were all normal, but auditory evoked potentials were abnormal.

His history prior to admission was extremely interesting and unfortunate. But it does illustrate some important factors.

He began working in the Maldives in April 1989. He usually dived twice a day, 6 days a week. The first dive in the morning was to 20 or 30 m for 50 to 60 minutes. The second dive was about 3 hours later and could be to 20 m. He used a Beuchat dive computer.

Occasionally he had the afternoon off. He drank heavily. He played hard. The staff at the resort noticed a slow personality change. He became aggressive and abusive, not only towards the staff but at times to the guests. He was told to stop drinking which he did, at least one month prior to his eviction from the island. His last recorded dive was on the morning of the 14th of September. He told me later that he does not know how he finished the dive. On ascent he became extremely confused. He remembered falling over in the boat. He was put on the first available flight back to Australia which was that day. He described the flight as being terrible. His confusion worsened. He disembarked at Singapore and stayed there for a week. His Darwin girlfriend reported receiving several distressing phone calls. She said his conversation was bizarre, vague and extremely slow. He finally arrived in Australia on the 22nd of September. He was unable to fill in his customs declaration form. He was strip searched by Customs as they suspected drugs. His girlfriend took him to Darwin Hospital. There he was admitted to the psychiatric ward. They were unable to diagnose the problem as his behaviour was unlike anything they had seen. Then finally somebody found out that he had been a diving instructor. They contacted us and he was flown to Adelaide that day.

It is of an interest that his employer sent all his possessions back to Australia except his dive log.

He received 10 hyperbaric treatments. He improved after each treatment, but even the optimists could never say that he was quite right. His auditory evoked potentials returned to normal.

He was reviewed on the 10th of October 1989. It was at this time that he had an acute paranoid psychotic reaction. He was hospitalized in a major psychiatric hospital. He underwent a series of investigations and psychometric assessments. Finally after rejecting our initial diagnosis the psychiatrists concluded that he had a "neuropsychological profile that one would expect from a person suffering from hypoxia, probable cause DCS". His presentation and what was subsequently found on psychometric assessment was not described in the text books.

Follow up revealed a pleasant fellow, off all medications and able to return to work. He still had a moderate degree of memory deficit. Assessment had also revealed frontal lobe impairment. However it is expected that he will make some spontaneous improvement.

Case 2

This 31 year old male.first dived 2 years ago, one of the dives was to 20 metres and involved a "slow" rapid

ascent. When he surfaced his symptoms were consistent with having suffered from a cerebral arterial gas embolism (CAGE) or cerebral DCS. He was told never to dive again. He waited for 2 years, then did four dives over 2 days. None were deeper than 15 metres. The bottom times were conservative and there were no instances of a rapid ascent. He surfaced from his last dive (to 10 m) with similar symptoms to his episode of 2 years earlier. He went home having decided to sweat it out. He slept that night with his bed on blocks to keep his head down. In the morning he was no better, in fact his symptoms were a little worse. Finally he contacted us. The diagnosis was made of cerebral DCS, with him probably having a right.to left shunt. (His daughter was diagnosed 2 days after his admission as having an ASD).

On admission he was extremely aggressive and abusive with paranoid overtones. He made remarks like "What are the police divers doing here? Are they after me?". He later said that he could not stop these odd feelings. He had a frontal headache, complained of paraesthesia and was very unsteady. There was short term memory loss.

His treatment was successful, to a degree!

Follow up revealed a different person to the one who had been admitted. He was a gentle caring father. He admitted to having had paranoid feelings, and also to having been extremely depressed. He said these feelings took about a month to disappear. He is at present undergoing psychometric assessment. He still complains of short term memory loss and of being "slower" than he was before this episode.

He will not be diving again.

Case 3

This Swedish tourist was treated at Townsville. She was reviewed at the RAH. She had been diagnosed as having had cerebral DCS. Although successfully treated clinically, she admitted to not feeling her normal self.

She was still slightly depressed, and said that this had gradually got better over the past couple of months. The interview also revealed that she had had extreme paranoid feelings during treatment and just after. At that time she did not like being alone because space creatures were going to get her. These creatures had been in a book that she had been reading when she went diving.

Discussion

Organic causes for depression and psychosis are numerous, although depression may just be a simple reaction to an environmental circumstance. Depression has some hypochondriacal associations. Some of the symptoms commonly seen after therapy, which disappear during hyperbaric treatment, may just be a manifestation of this depressive phase. Common symptoms frequently seen are poorly localized joint pain, insomnia, fleeting nondescript pains, fatigue and generally feeling unwell. Perhaps we should be using antidepressive medication in some of these patients, especially if the symptoms are different before and after treatment.

Encephalopathies following organic illnesses are numerous, e.g. delayed hypoxic encephalopathy is characterized by apathy, confusion, irritability, agitation and mania. Endocrine encephalopathies are characterized by confusion, agitation, hallucinations, delusions, anxiety and depression. But perhaps the one clinically closest to what I have described here is puerperal psychosis. This is a brief psychological disturbance seen in the puerperium. Typically it is one of depression, however, there are other degrees of confusion and thought disorders. Recovery takes time.

The study by Adkisson et al[1] showed that the cerebral perfusion deficits in divers suffering from Type II DCS were mainly in the frontal and parietal lobes. The clinical significance of this was unclear. Perhaps it may manifest itself as depression and/or other psychiatric or psychological problems. Psychometric assessment of one of these cases showed frontal lobe involvement. Adkisson et al. described that their divers had disorders of mentation, but what these disorders were was not described.

Conclusions

I have presented reflections based on clinical observations. To be bent and not recognize it may in fact be an important diagnostic tool for cerebral DCS.

The symptomatology of DCS, which is obvious to us may not be of importance to the diver because the various pathological processes invovled alter perception. Reluctance to seek treatment may actually be part of the disease process due to specific CNS deficits. Indeed aggression, abnormal and paranoid behaviour indicate significant CNS involvement.

A good clinical guide to a diver's well-being after DCS would be the diver's immediate family's assessment of when the diver is back to "normal" again. Unfortunately this may never occur.

Reference

1 Adkisson GH, MacLeod, Hodgson M et al. Cerebral perfusion deficits in dysbaric illness. *Lancet* 1989; ii: 119-122

This is an editorially revised transcript of a lecture given at the 1990 Annual Scientific Meeting. Full references

are available from Dr Acott.

Dr. Chris Acott FFARACS is attached to the Hyperbaric Medicine Unit, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia, 5000.

A SERIES OF DIVING ACCIDENTS FROM NORTHLAND, NEW ZEALAND, 1984 - 1989

Peter Chapman-Smith

Introduction

This group of 80 cases was collected in a region of northern New Zealand, where snorkel and scuba diving is a prevalent leisure and food gathering activity. Most cases presented in general practice, by referral from dive shops or from the local Base Hospital in Whangarei.

An overview is provided, followed by consideration of several specific cases of inner ear barotrauma and then some unusual cases.

Clinical Material

80 cases presented over this 5 year period.

BAROTRAUMA

Ear	
External	1
Middle	17
Ruptured tympanic membrane	8
Alternobaric vertigo	2
Round window rupture	11
Inner	3
Sinuses	

Although it is my impression that this is the most common problem only 1 case was recorded.

PULMONARY	
Arterial gas embolism	
Cerebral (CAGE)	9
Mesenteric AGE	1
Pneumothorax	1
Other	2

DECOMPRESSION SICKNESS (DCS)

Of the 8 cases none had neurological DCS.

Skin	1
Elbow	2
Knee	1
Shoulder	3
Gastro-intestinal	1

TRAUMA

This group was quite varied. The most dramatic was the case of traumatic pneumothorax. The others were:

Neuropraxia of ulnar nerve	1
Head injury	1
Foreign bodies in the skin	2
Subconjunctival haemorrhage	1

MISCELLANEOUS

Shallow water blackout	2
Dermatitis due to jellyfish contact	4
Near drowning	2
Retinal haemorrhage	1
Hypoglycaemia in an insulin dependant diabetic.	1

Inner Ear Barotrauma

The cases of inner ear barotrauma were of interest. To differentiate between the round and oval window ruptures, pure cochlear or vestibular damage, or inner ear membrane rupture can be difficult. And there is always the possibility of inner ear haemorrhage. Even at tympanotomy a demonstrable fistula is often not apparent.

PRESENTATION

Of the 14 cases, 2 were snorkelling, the remainder were scuba divers. There were equal numbers of left and right round window ruptures.

Seven divers had considerable dive experience Nine had had difficulty during the dive performing an adequate Valsalva manoeuvre. Nine developed symptoms while in the water Symptoms were usually of sudden onset. Eleven divers complained of deafness. Giddiness or vertigo was noticed by five. Ten had tinnitus and it was often still present when they were seen. Four had nausea which was often marked, while four vomited. Other symptoms reported were epistaxis, full or blocked or watery sensation in the ear, hyperacusis, and pain. Hyperacusis, especially acute hearing, was an unexpected finding. Edmonds[1] mentions that some patients with a patulous Eustachian tube, following barotrauma, may have excessive awareneess of their respiration or of their own speech (causing them to speak softly) or reverberations of sounds such as footsteps. My patients did not complain of these problems.

Three divers had been using treatment for allergic rhinitis. The divers were frequently late in presenting, appearing days or weeks after the day of the dive accident. They had often seen an after hours GP, casualty officer or

other doctors. The history took some digging out on occasions as the patients were reluctant to complain.

DIVE HISTORY

Most had been diving in less than 18 msw. However, 1 diver had descended to 60 msw. Frequent ascents and descents were a common feature of the dives.

TREATMENT

3 divers were operated on, with tympanotomy and the application of a fascial graft. Most received either conservative or no treatment. Rest, head up posture, carbogen (CO_2), oxpentifylline, avoiding straining and tranquillizers were tried for some. Most of the cases, being late presenters, received no useful therapy. Steroids were tried in 1 case of inner ear membrane rupture.

OUTCOME

Constant tinnitus was a feature in this group, with permanent significant sensorineural deafness on the affected side, principally in the high frequencies, usually > 4,000 - 6,000 Hz.

Two were left with less than 40 dB hearing loss, both having had tympanotomy and repair. One diver with inner ear barotrauma was left with a 40 dB low frequency loss. Two had moderate (40-60 dB) high frequency loss. Six had severe (more than >60 dB) high frequency hearing loss. One of these had a 50-60 dB loss above 2,000 Hz. Another had a loss in the left ear of 60 dB above 4,000 Hz and 70 dB above 6,000 Hz in the right ear. One of the snorkellers, who presented the same day, had a 120 dB loss above 4,000 Hz.

Three patients were lost to follow up.

Goodhill[2] described one way a round window fistula occurs. The cochlear aqueduct communicating between the cerebro-spinal fluid (CSF) and perilymph is of variable structure and calibre. If a short duct fails to damp a pressure wave from the CSF, the sharp rise in pressure in the scala tympani could cause a window rupture. The round window membrane is only 3 layers of 4 or 5 cells thick and of variable thickness, 10 µ centrally to 70 µ peripherally. Alternatively, the pressure wave could rupture through the basilar and Reissner's membranes to involve the utricle and saccule. A further mechanism is excessive rapid inward or outward movement of the ear drum. The first raising the inner ear pressure by pushing the ossicles in and the second, by suddenly pulling on the ossicles in the oval window, dragging the round window inwards beyond its tolerance. This explains why inner ear barotrauma can be associated with sudden middle ear barotrauma or a forceful Valsalva manoeuvre.

I have made no attempt to consider the possible causes of sensorineural hearing loss and vestibular symptoms as seen in DCS or inner ear haemorrhage on ascent. No doubt some "occupational hearing loss" cases seen in general practice stem from these causes.

These cases are only those that were recognized. I suspect there are in fact many more that pass unrecognized. The preponderance of middle ear barotrauma is as expected. The extremely common sinus barotrauma and cases of otitis externa have not been shown in this study in their true numbers because they are so common, and were not recorded.

The new data on right to left cardiac shunts with potentially patent interatrial septa, suggests that inner ear air embolism may be more common than previously considered. As exact diagnosis is not easy and with their late presentations, one can only urge medical colleagues to take an often lengthy history, to use an auriscope, to use an audiometer and to try to identify these patients with inner ear barotrauma, who can do so well with early appropriate treatment.

Case 1

A 26 year old farmer presented in February 1990 with a story suggestive of bilateral round window rupture. He had been diving 2 or 3 times a week, using a 5 mm wetsuit and hood, for 3 years. He first had problems diving in a fresh water lake in Northland. In 1984, experiencing difficulty clearing his ears underwater, he developed a sudden pain in his left ear. He felt generally unwell for a week after. He recalled imbalance and falling over to one side.

Some 3 years later, whilst cray hunting, he swam hard on scuba to a cave at 6 m for about 30 minutes. Feeling well he had a 45 minute surface interval. The next dive was to 12 m, with a number of descents and ascents. 15 minutes into the dive he felt unwell with marked nausea and vertigo. Surfacing, he felt quite giddy, was vertiginous and vomited a lot. He remembers a yellow mucoid post-nasal drip after the dive. On closer questioning, he admitted to recurrent episodes of vertigo, giddiness, nausea and vomiting underwater. He had had approximately 12 attacks over the previous 2 years. The onset of symptoms was usually when diving in a swell or near the surface. At presentation he felt he was deaf bilaterally. He clearly recalled the two episodes of acute onset pain before the vestibular symptoms being on opposite sides.

His past medical history was not abnormal. He took no regular drugs. But he had some allergic rhinitis. Occupational exposure to noise had occurred.

On examination, apart from minor otitis externa, only a severe bilateral high frequency hearing loss was noted, 60 dB above 4,000 Hz in the left ear and 70 dB above 6,000 Hz in the right.

The diagnosis was presumed bilateral round window rupture.

Case 2

This 28 year old worker presented 1 month after diving in the sea at Taupo Bay on the Northland coast in 1987. He recalled a day with a lot of surge. He had scuba dived to 10-12 m and suddenly developed nausea and vertigo underwater, vomiting several times. He experienced tinnitus and a dull feeling in his right ear. He often had problems equalizing. Examination showed a serous middle ear effusion and an 85 dB loss at 6,000 Hz.

Following admission to the local base hospital, there was no improvement in his serial audiograms and he was taken to theatre three days later. At tympanotomy, a round window rupture was noted and the fistula was patched. He had a splendid result with only a 35 dB loss in that ear and no vertigo, however some tinnitus still persists.

Case 3

A 22 year old ticketed scuba diver of 1 year's standing, who had had no prior problems with his ears went spearfishing in the Whangaruru Harbour region in January 1989. Snorkel diving to recover a spear, he developed considerable vertigo when back on the surface. Climbing out onto the nearby rocks, he noted nausea, left sided deafness and tinnitus. These symptoms persisted for 5-6 hours. At that time his balance was poor.

The next day he had a planned scuba dive lined up. The first scuba dive was to 12 m chasing crays and 45 minutes went by without problems. After a surface interval of 20 minutes, they dived again to 12 m. He developed vertigo in the water with marked nausea and surfaced feeling unwell. Having lost his buddy, he dived again to seek him! Not locating the other diver and feeling worse, he surfaced and vomited in the boat a number of times. Marked vertigo continued.

Hewent to the local hospital casualty room. Next day he still had nausea, giddiness, deafness and vertigo. His audiogram showed a 60 dB loss above 6,000 Hz. The local consultant was away he was sent to hospital in Auckland. Although he had presented acutely his round window rupture with a perilymph fistula, was treatment conservatively with rest, carbogen (CO_2), oxpentifylline, and steroids.. His audiogram never improved, although his vestibular symptoms were reduced. He was given a hearing aid and advised that to resume diving in 2 months would be safe !

Case 4

This 33 year old, experienced diver presented in December 1989 after scuba diving with a friend looking for crays. During the first descent he had some difficulty in clearing his ears. The dive profile of the first dive was to 18 m for 20 minutes, followed by a surface interval of 30 seconds. A second descent to 18 m for 20 minutes. He then surfaced again for 1 minute as his buddy had lost his weight belt. Then a further descent to 18 m for just 5 minutes. Then there was a surface interval of 50 minutes.

The second dive was to 18 m for 25 minutes, then he slowly ascended to spend sometime at 9 m and to do a 6 minute stop at 3 m depth. Following the second dive he noticed a left hearing loss, but no vestibular symptoms such as nausea, vertigo or tinnitus. The ear felt blocked.

He sought advice from the on-call GP and was prescribed an antihistamine. He presented to his usual GP ten days afterwards. A middle ear effusion was noted with a negative Valsalva manoeuvre on the left, poor mobility of the tympanic membrane and an apparent conductive hearing loss. In fact a severe high frequency loss was present, 75 dB above 6,000 Hz in the left ear. There was minimal improvement 1 month later, after a course of prednisone, which was suggested by the local ENT consultant, for presumed pure inner ear barotrauma. His hearing loss appears to be permanent.

Case 5

A 41 year old professional man presented acutely in January 1989 after scuba diving for scallops in the Bay of Islands. There was no significant past medical history. Trained 3 years previously, he dived regularly.

The first dive involved many descents and ascents to 7.5 m over 45 minutes. His catch bag was attached to his buoyancy compensator. Developing cramps in his legs, he started to feel generally mildly unwell. On his last dive he coughed, ingesting compressed air from his regulator. On surfacing he felt quite unwell and noted the sudden onset of left upper abdominal pain. He snorkelled 70 m to the waiting yacht. Clambering aboard, his abdomen felt distended. Passing urine and having a bowel motion slightly relieved his discomfort. He then vomited, lay down, and was taken to the wharf at Russell.

Here he was very well received and resuscitated by the local GP and his nurse. He was placed in the left Trendelenberg position and given oxygen. Advice was sought over the phone. He complained of back pain and left upper quadrant abdominal pain and paraesthesiae and mus-

cle twitching in his lower legs.

He was transferred by helicopter to Whangarei. Here he was further assessed. He was pale and clammy, but alert and orientated, with an irregular pulse. His cardio-vascular system was otherwise stable with good cardiac output and respiratory status. Arterial blood gases showed a PO_2 of 250 mm Hg (on 6 l/min O_2), PCO_2 of 52.9 mm Hg, otherwise they were normal. An ECG and chest X-ray were normal. An intravenous drip was inserted and he was sent on to RNZN Hospital in Auckland for recompression.

In transit, despite 30 mg of papaveretum given intramuscularly he was in constant discomfort, rubbing his abdomen and some shakiness of his left leg continued. Treated with a table 6A in the naval recompression chambers, his symptoms resolved readily under pressure. He was neurologically normal the next day and was sent home symptom free. The differential diagnosis included aerophagy, mesenteric arterial gas embolism, free peritoneal gas or a combination of these.

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1 Edmonds C, Lowry CJ. and Pennefather J. *Diving and subaquatic medicine*. 2nd edition revised. Sydney: Diving Medical Centre, 1983 392

2 Goodhill V. Sudden deafness and round window rupture. *Laryngoscope* 1971; 81: 1462-1474

This is an edited transcript of a lecture given at the 1990 Annual Scientific Meeting of SPUMS.

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THE DEVELOPMENT OF THE RECREATIONAL DIVE PLANNER

Ray Rogers

Summary:

Depth increments in the US Navy tables are too great. The repetitive dive table is based on the slow-responding 120 minute tissue compartment, so little surface credit was obtained. This compartment was largely irrelevant to recreational diving. The repetitive group format was unsuitable as the time/depth benefits were unharmonious and times were excessively rounded off, creating anomalies. Research suggested lowering of no-stop limits. It become apparent that wholly new tables were appropriate as the USN tables were too "coarse" were not planned for extensive repetitive diving and the USN tables do not permit multi-level diving.

The compartment structure seemed wrong as there were too few compartments and they were internally inconsistent. I added compartments and adjusted their values. The 120 minute compartment never seemed to have an effect while the 40 minute tissue usually controlled the dives. The 60 minute tissue occasionally controlled and was chosen for controlling the repetitive dive calculation. The resulting table was more conservative and the time penalties were not great. The 120 minute compartment is important in long, deep dives with staged decompression.

The basic concept of theoretical model was Haldanian, retaining exponential gas exchange and a spectrum of tissue compartments. The modifications were variable maximum allowable tissue pressures, an increased number of compartments, Hempleman's power function used for the non-stop curve, shallow and deep asymptotes added, the no-stop curve was smoothed and faired, "M-values" were derived from this curve, discontinuities were eliminated and no-stop limits were lowered.

The table was designed as a circular slide rule in polar format. Multi-level capability was included. The procedure is that the dive always goes from deep to shallow. Adjustments were made to keep pressures within limits by minimum depth differentials on ascents and time restrictions were added to no-stop limits. Safety stops at 15ft/5m were recommended after all dives. These stops are required after some dives, those deeper than 100ft/30m and when within 3 pressure groups of any limit. The advantages of stops are: a dramatic reduction of tissue pressures, compensation for staying too long, compensation for diving too deep, compensation for gauge or timer error, and a slower ascent rate. There are special rules which require long surface intervals occasionally. These are when pressure groups become very high, as after repeated long, shallow dives. The rules are seldom required, but they exist.

Introduction

It is a great pleasure to be able to speak to SPUMS about the development of the Recreational Dive Planner, so enthusiastically and overwhelmingly embraced by over 95% of the medical community Down Under. Unfortunately those people have been so struck dumb by the brilliance of it all that they have remained absolutely mute and have not been able to comment. But the five per cent who are not excessively enthusiastic have written horrible letters and and numerous articles. So I would ask that they briefly give a few moments of their attention to this talk about the development of the Recreational Dive Planner (RDP) and the corporation that was created to develop it, Diving Sci-

ence and Technology (DSAT). The Recreational Dive Planner comes in a rotary format, the PADI Wheel, and also in conventional tabular format.

The need for new tables

The RDP has filled a clear and obvious need for a new table. I suggest that the need for new tables is self evident, witness the BS-AC tables and DCIEM tables. Also there are many different types of decompression computers. If the US Navy Tables were ideal, presumably these new tables would not be needed. Presumably the network of recompression chambers around the world would not exist, because there would be no need for them either.

Everyone has his own private list of why the US Navy Tables should be redone. Here is my list:

Depth increments too large

First of all, I think the depth increments are entirely too great. Depth increments are in 5 foot steps from thirty to forty feet, and thereafter ten feet, (Bear with me, Australians think in imperial units much better than I think in metric, so will be imperial all the way). What I call coarseness is a large decrease in available time with a small increase in depth. Between forty and fifty feet bottom time drops from 200 minutes to 100 minutes, entirely too big a step in my opinion.

Need for shorter no-stop limits

Research has suggested that there should be lower no-stop limits. During the 70s and 80s many people investigated the application of Doppler technology to diving, but it was Merrill Spencer[1] who first suggested that, for recreational use by the average diver, no-stop limits in the US Navy tables were a bit high.

If one simply wanted to reduce no-stop limits, it would be easy to take a waterproof marker to the old tables and mark the new limits on them. In fact, this has been recommended in an article in the Undersea Journal, PADI's house organ.[2]

Repetitive diving table problems

There are other issues such as the repetitive dive table being based on the very slow responding 120 minute half-time theoretical compartment. I will refer to it as a compartment or tissue. If a 120 minute half-time tissue is exposed to a pressure gradient, after two hours it has taken up to half the gas load that it can take up. It will take a further two hours take up half the remaining potential gas load. In other words these tissues have exponential uptake of gas. Since most recreational dives encompass a much shorter time span , what is the relevance of the 120 minute tissue?

In the PADI version of the USN tables ,for recreational divers, at the top of table two, is printed "surface interval credit table". In my opinion it should be the surface interval "no-credit" table, because sometimes after approximately 45 minutes one is in exactly the same pressure groups as one started in; one gets no credit at all. If one happens to be in groups E through H and on the surface for something like an hour or an hour and fifteen minutes, which is very common in recreational diving, there is a gain of one group, because the groups are not small enough. When the USN originally calculated the the repetitive dive table, they had a total of 31 pressure groups, differing by the equivalent to one foot of seawater absolute. But to simplify things, they grouped them together into two feet of seawater pressure groups. So we are burdened by a broad pressure range secured by a very slowly responding tissue.

If one looks at the residual nitrogen times one sees that at one depth the time gained by the diver as he is off gassing, going from pressure group to the next, on the surface is quite small. But as one goes vertically, from depth to depth for the next dive, the increments are very large. That is what I mean by coarseness. The groups are too large and the surface interval credit table is quite unresponsive. The tables were rounded off excessively, and created many internal anomalies. The residual nitrogen time table was calculated to the nearest minute, while the front part of the table was calculated and rounded off to the nearest five or ten minutes. This was originally based on the Navy Experimental Diving Unit (NEDU) reports in 1955-1957[3-6] which recommended that it should have been recalculated to the nearest minute. This was never done, and as a consequence there are a lot of anomalies.

My personal favourite, which combines all these discrepancies, is this example. If I am on a repetitive dive and my total bottom time is 141 minutes, and I have been at exactly 40 feet for this entire time, all the tables grant me virtually another hour of bottom time. But if I descend momentarily below 40 feet, say to 41, I am now under a decompression obligation. I find this quite unreasonable. I have asked a lot of people why it should be this way, and they say, "That's the Navy table". But I am not in the Navy and I do not care. I tried to find an answer, but nobody knew why. Nowadays, I think a lot of people know, but even five years ago knowledge was very sparse on the subject.

Reworking the USN tables.

Luckily I found a book written about 12 years ago[7] and there was information that led to the papers[3-6] which were used to set up the original Navy tables 35 years ago, and

by working my way through them I finally figured out what the Navy did when it created these tables.

I made copies of the worksheets. All the calculations were done by hand. One should try it sometime, without a calculator or computer. It may take several minutes to figure out one tissue pressure for one exposure and one compartment, and then one has to do it all over again. It takes a long, long time and it is very tedious.

In the process of doing these many, many calculations, I found to my great surprise that the 120 minute compartment was not relevant. By then I knew that these tables were based on the 120 minute tissue. But I computed typical recreational profiles (reading from my old log books for what I had actually done) and the controlling compartment was always much faster. It was typically the 40 minute compartment and I did not understand that. Along the way I noticed that there were many discontinuities in the graphs I drew. This was because there were too few tissues in the model, so I added a few of my own, 30 minutes and a 60 minutes which the US Navy had never done. They went on a scheme of 5,10,20,40,80, 120. I programmed a 30, a 60, a 90 and 100 and found that this redundancy of tissues was really quite useless because it splits a hair too fine. But adding 30 and 60 minute tissues happened to fill big gaps between the 20, 40 and 80 minute tissues. I postulated a series of dives that were long and shallow, repeated over a number of days. Once in a while the new 60 minute compartment which I had added would be the one that would reach this limit and go over. I decided that since this could apply on some occasions, we should make this the tissue of choice for calcualtion of surface interval credit tables.

Recreational diving and the 120 minute tissue

Early in 1990 I attended the meeting of the Gulf Coast Chapter of the UHMS. Some graduate students were presenting a paper about electronic computers and comparing them to the US Navy tables. They went on and on with table after table of theoretical dives and kept talking about omitted decompression time in computers, implying that the US Navy table was still, somehow, the gold standard. What they could have said equally well, was that according to the computers, the US Navy required a whole lot of unnecessary decompression time.

In Figure 1 there is a simple series of dives of 60 feet for 30 minutes, followed by a relatively generous 75 minutes at surface, a repeat of 60 feet for 30 minutes, repeat the surface interval, and repeat the dive. Across the top is the US Navy M-value or limiting pressure, the maximum allowable pressure in the 120 minute tissue permitting direct ascent to the surface. The lower line represents the actual generation of pressures in the 120 minutes compartment. At the end the dive it is about half way up to the maximum allowable pressure, and yet the tables would require about 14 minutes of decompression.

In Figure 2 the curve at the top is the combination of

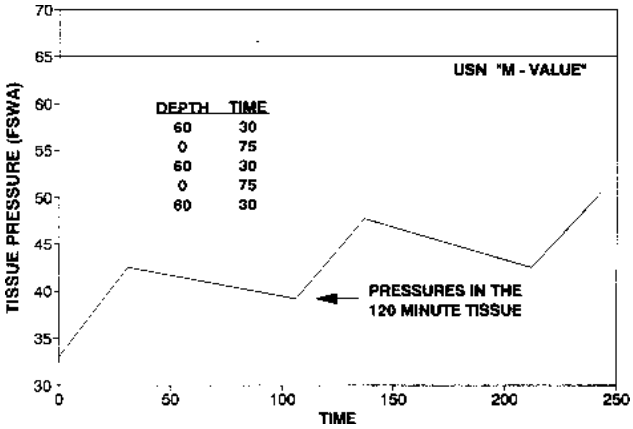

FIGURE 1

Pressures in the 120 minute compartment for a series of three dives to 18 m (60 feet) for 30 minutes with a surface interval of 75 minutes after the first and second dives.

depths and times which are required to generate the maximum pressure in the 120 minutes tissue compartment, that the US Navy defines as permitting direct ascent to the surface. The irregular lower curve is the actual plot of the US Navy no-stop limits. There is a great gap.

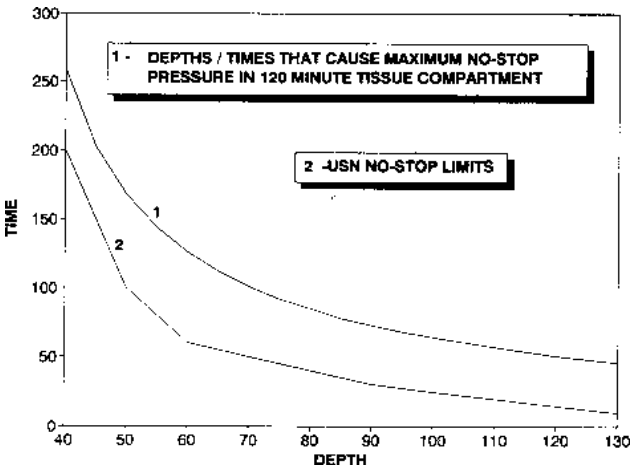

FIGURE 2

Comparison between depth/time curves, that which causes maximum no-stop pressure in the 120 minute compartmen and the USN no-stop limits.

Table 1 shows the same information for various depths. The second column, 120 max, is the number of minutes at those depths required to pump up the 120 minute

TABLE 1

TIME AND DEPTH COMPARISONS OF THE 120 MINUTE HALF-TIME TISSUE MAXIMUM AND THE USN AND DSAT NO-STOP LIMITS

Depth	120 max	USN	DSAT
40	259	200	140
60	126	60	55
80	85	40	30
100	64	25	20
120	51	15	13

tissue to its maximum allowable level and the USN no-stop limits (NDLs) are in the next column while the DSAT NDLs are in the right hand column.

One could stay for 64 minutes at 100 feet and still be the 120 minute tissue would be at a lesser pressure than the Navy says is acceptable, but the USN NDL is 25. The Recreational Dive Planner NDL is even more conservative, just a tiny little portion of the M-value. Just because something has been around for a long time does not mean that it is relevant.

Navy diving is different

Why did the USN use that compartment if it is so insignificant? Well, it was not insignificant for the US Navy, which was planning for deep, long dives such as 100 feet for two hours or 180 feet for one hour to be followed by repetitive diving. It is very significant under those conditions. But we do not do that sort of thing in the recreational world. The Navy was trying to make tables that would be able to extend working times, or bottom times, while decreasing the total amount of decompression time required, all the way to 300 feet. The idea of creating a table which might later be of use to people like us, who go out to look at pretty reefs, was the furthest thing in the world from their minds.

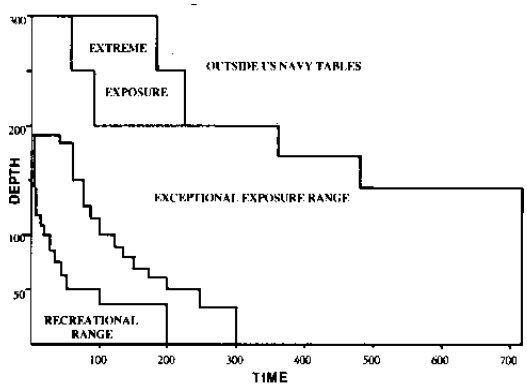

FIGURE 3

The depth time relationships of the USN tables.

Figure 3 shows graphically what I am talking about. The entire span of the Navy tables is 300 feet of depth and 720 minutes, that is 12 hours. The section down on the lower left is that part of the overall decompression tables which recreational divers, who came along some years later, borrowed from the Navy Tables, reformatted it, and called it the Navy Tables. The Navy did not mind. We only dive in the tiny bit in the corner.

We do not do salvage work or underwater demolition. What we do is look at the pretty fishes and enjoy the scenery. And for that we need something for ourselves. I am not suggesting for a minute that these tables are wrong, but just that for the diving we do they are just not appropriate.

Principles for recreational diver tables

To achieve appropriate tables we made yet another modification of the method of calculating decompression schedules, which was first developed by J. S. Haldane early in this century. Seldom has the work of a single person dominated a field for eight decades and stood the test of time so well. Virtually all tables and dive computers in existence today use some type of adaptation of Haldane's methods. It has become fashionable to deride Haldane, even as his critics continue to employ his procedures. I realize that many of his premises are considered invalid today and that there are a lot of things he did that people do not accept any more, but the man's work was brilliant. There are two things that are customarily retained from the original Haldanian algorithm, the concepts of exponential on-gassing and off-gassing and the spectrum of tissue compartments.

Through the years, there have been many adjustments to Haldane's work and current tables seem to bear little similarity to those he created in 1908. Every revision changed an earlier version to accommodate new data , and certainly, the RDP was no exception, imposing once again a great deal of modification.

This equation, $D=500\sqrt{T}$ is a variation of Val Hempleman's $Q=P\sqrt{t}$, wherein Q is a fixed quantity of dissolved gas, P is depth and t is time. In the variation, D is depth, T is time, and 500 is a derived constant which retrofits the US Navy no-stop limits to the equation. As simplistic as this may seem, the fit is generally quite good, and it is largely correct. This can be generalized to the form $D= CT^x$, which says that depth and no-stop time bear an inverse exponential relationship as described by the constants C and x (which are derived from any given table by using any two no-stop limits from the table). The equation may be rewritten in the form $D_1T_1^x = D_2T_2^x$, which suggests theoretically at least, that within the given framework of limited exposures of no-stop diving, one dive is exactly equal to another in terms of decompression stress. That may not true, but if we go with that premise we can get an advantage out of it.

Graphing the no-stop limits on a linear scale gives a sweeping curve, and if one wants to compare tables, it is difficult, because the lines are curved so much that it shows nothing. Spencer[1] showed that an exponential curve plotted exponentially or logarithmically is a straight line. This allows rapid comparison between tables. By putting one straight line on another it is easy to see which is more conservative.

The equation can work for any two observations of time and depth. One gets, in the normal recreational depth range, a remarkably good fit of my calculations with the USN NDLs. However at the extremes the accuracy falls off sharply. The formula suggests that one can do non-stop diving to 400 feet and that is not true. Even if you did not stay for any time at this depth, you would still be forced to do a decompression stop. At the other end no one is going to stay for three weeks at 10 feet. As most people ignore these extremes and stay mainly in the middle, the calculation is useful.

The anomalies bothered me. It was simple enough to correct for the error at the shallow end of the curve. One can assume that there is some shallow depth that one can go to virtually indefinitely and make a direct ascent to the surface. One can argue about what that depth is. At this depth, bottom time becomes infinite, and the no-stop limit curve becomes asymptotic with the depth. If this asymptote is added Hempleman's equation becomes ($D-A=CT^{-x}$)

A diver accumulates nitrogen all the way down and most of the way up, and there is a point at which the time spent in descending and ascending to a certain depth will equal the theoretical limit of the model, even if the diver did nothing but turn around for an immediate ascent. This calculated depth happens to be 243 feet in the DSAT model, which is obviously far beyond the permitted depth of 130 feet, but it is useful in the calculation and plotting of the no-stop curve of the DSAT algorithm. Figure 4 shows the DSAT NDL curve with the original straight line. One can see that a no-stop limit between the marks is a little more conservative than the straight line, but more obviously, the unnecessary deficiencies of the $D=CT^{-x}$ have been eliminated. At this point, we can dispense with the straight line and consider only the sigmoidal curve which reflects more realistically what no-stop limits probably are.

Development

Instead of starting with a whole series of empirical observations of time and depth and saying and draw a no-stop curve; I started the other way. This makes more sense intuitively, and if I could make empirical data match the curve at a few points, then I could start deriving a lot of data from the curve; which is exactly what I did.

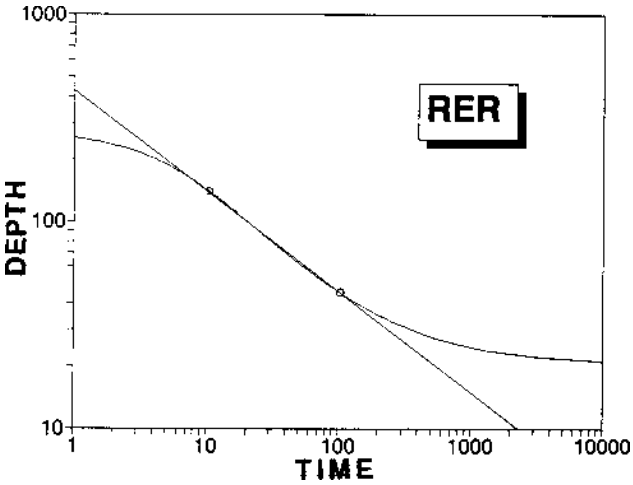

FIGURE 4

The original Rogers no-stop curve plotted, as a straight line, with the modifications needed to remove unacceptable figures (curve) superimposed.

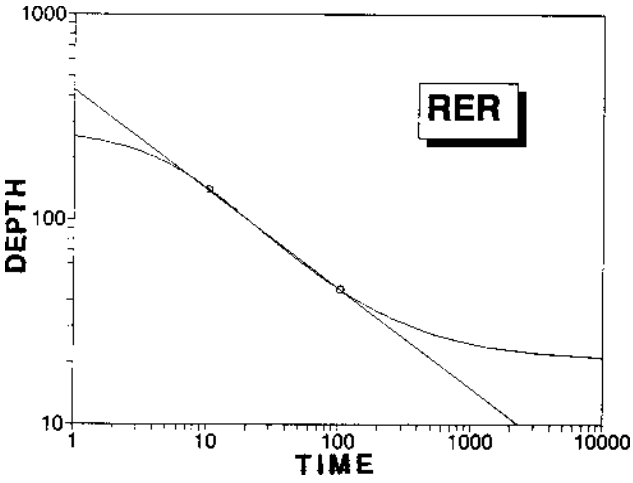

FIGURE 5

The modified Rogers curve with the RDP no-stop limits superimposed.

Figure 5 shows the no-stop limit of the RDP placed over over my no-stop curve. The little tail that sticks out at the top left is foot by foot calculations of the no-stop limits, which go all the way down the line. But one does not see them as a separate curve, because they superimpose perfectly. They superimpose because the no-stop values were derived from the empirically generated curve. I started with the no-stop limits and found a formula to predict them accurately.

Figure 6 shows the same thing done with the USN no-stop limits. They are all over the place, up and down, left and right. It is not reasonable to accept this. Intuition suggests that these irregularities and discontinuities would not exist in the water column, which is a smooth gradient

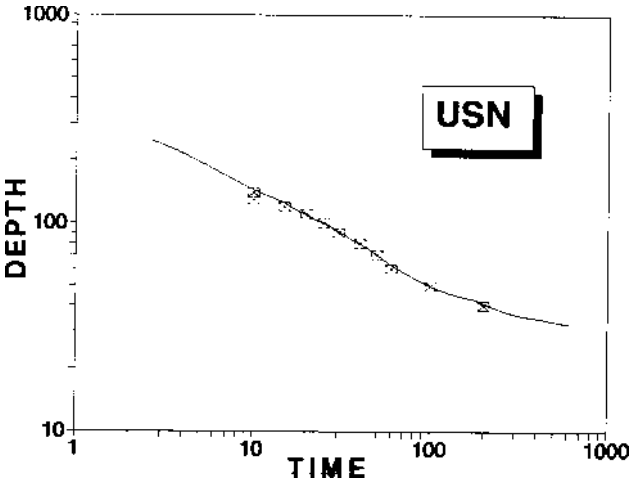

FIGURE 6

The USN curve with the USN no-stop limits superimposed.

increasing or decreasing depending on the direction one goes. I have used this concept of continuity to evaluate various other tables that are around. Take a look at them, and one finds some remarkable problems with some of the tables.

I suggest that tables should have internal consistency. I think that is important. However, every other system that I have seen, that is commercially available today anywhere worldwide, is full of internal inconsistencies.

Our tables are in based on the principle of consistency. We built them for recreational divers. Figure 7 shows that the no-stop limits which apply to the first dive of the RDP are more conservative than the USN NDLs. RDP limits

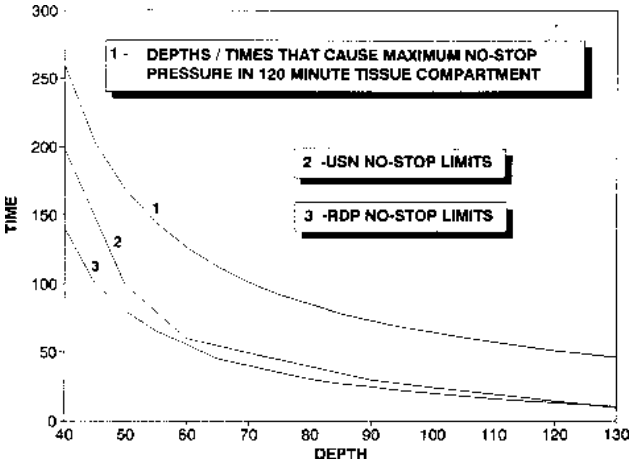

FIGURE 7

Depth/time curves showing (1) maximum no-stop pressures in the 120 minute compartment, (2) USN no-stop limits and (3) RDP no-stop limits, both for a first dive.

were taken from the curve which was developed the way as I have described.

The Wheel format

Along the way, a notion occurred to me. Instead of displaying a few episodic solutions to the whole equation for various combinations of times and depths over the place, why not use the curve itself and get rid of the rows of numbers that are found in typical tables. The RDP Wheel uses only time and depth, the only things one actually measures.

There are significant changes between the RDP table and most of the standard tables. One thing was five foot increments, which breaks up the problem of coarseness. Another, is that the RDP had multi-level diving built in from the very outset. The flat version of the RDP is basically the same as the Wheel in the information that it presents, but the Wheel presents a lot more information.

Multi-level diving

Multi-level diving is desired by a lot of divers. Multi-level diving has been part of the RDP from the outset. It is the only system that I am aware of that has been tested.

Multi-level diving is not new. These techniques have been around for 15 to 20 years One does it with existing tables by "sliding sideways" through repetitive groups to get an equivalent pressure group for a later level. But are only attempts to adjust a model that was never designed for it. Multi-level diving is quite practical, if the model is altered to allow shifting of a pressure group from greater to lesser depths. Multi-evel dives are appreciably different from "square dives". If one stops part way to the surface, it is typical that the tissue compartment which first reaches a limit is "faster" than the compartment that usually "controls" the depth, and any method that promotes such diving must provide for this occurrence and be cognizant of precisely which of many compartments is nearing its maximum tolerable pressure.

There is no table in the world today which is based on a single tissue compartment, whatever you might have heard. It is said that the US Navy tables are based on a 120 minute tissue compartment. That is only true of the surface interval credit table. It has been said that the Wheel is a single tissue model which only uses the 60 minute compartment and ignores all slower compartments. Well, people who say you that probably do not know about other things too.

No table can get away with that. The no-stop limits confirms that fact and as depths increase, the repetitive

groups of the no-stop limits increase progressively. When PADI formulated its version of the old Navy Tables, it marked off its no-stop limits in black.

There are many other compartments are in the US Navy model and PADI did not want the diver going past the no-stop limits if one is doing no-stop diving.

The no-stop limits do not mean that one has gone over the limit in the 120 minute compartment. But one may have gone to the limit in the faster compartments. The no-stop limits for the greater depths are determined by the 5 minute compartment, ranging down to the 80 minute for the shallower depths. The compartments that control recreational diving range from 5 to 80 minutes when using the USN table. The 120 minute compartment plays no part, except as calculations of pressure in that compartment for use with the surface interval credit table.

Modifications required for multilevel diving

Multi-level diving is affected by many pressure groups and adjustments have to be made to stay within the limits of all the model's compartments, no matter what kind of crazy profile one may choose to adopt.

Figure 8 is a graph of two simple square dives, plotting the theoretical effect in eight theoretical compartments, and one can see the dynamic interplay of all these tissue pressures through this series of dives. If it is proposed to do multi-level diving as well, it becomes a virtual impossibility to allow for all the complexities one has to start with a series of simplifications to even begin the process. It does not take or too many calculations to make a couple of simplifications.

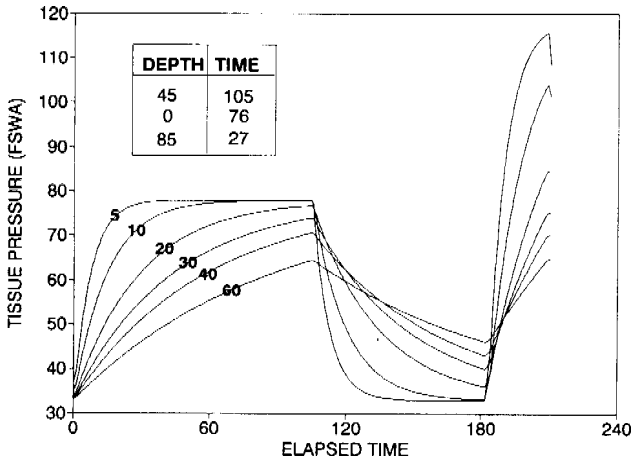

FIGURE 8

Tissue tensions in various compartments during two dives. The first dive is to 13.6 m (45 feet) for 105 minutes followed by a surface interval of 76 minutes with the second dive to 25.8 m (85 feet) for 27 minutes.

First, the permitted time at the second level is least if the depth at the first level is very close to the second. Secondly time is least at the second level if one goes to the non-stop limit at the first level. And if one puts these two together and makes a set of adjustmentsone has allowed for the worst case and all other possibilities are conservative.

We established a series of minimum depth differentials. We took groups of three depths and lumped them together into what we call a range of first depths, for example 140 feet through 120 feet make one depth range. We calculated what would be the maximum permitted depth for the second level, in this case 80 feet if one is in that first depth range. Table 2 shows the ranges and maximum depths of the second level. If one dives one's first level between 90 and 80 feet, one needs to go at least as shallow as 60 feet to be able to define it as a multi-level dive. Otherwise the old rule of total time at the maximum depth still applies. But if one can get a sufficient differential of depth then it can be called multi-level.

TABLE 2

RDP RANGE OF FIRST DEPTHS AND MAXIMUM DEPTHS OF SECOND LEVEL OF MULTI-LEVEL DIVES

Range of first depths			Maximum depths of second level
140	-	120	80
110	-	95	70
90	-	80	60
75	-	65	50
60	-	50	40

Step number two is to calculate the multi-level time adjustment. One does that by substracting the no-stop limit at the shallowest depth in one of these groups of three, from the no-stop limit of the second depth that would be permitted from the first depth range. This reduces the no-stop limit on each depth curve in such a way that no time does one ever go over the limit.

On the Wheel any curve out at the no-stop limit, marked as NDL, would be the normal amount of time permitted for a single level dive. But back up the curve of time is the multi-level limit, which says if one is doing this from deeper depth, and this is the second or third level of a multi-level dive, one is allowed a lesser amount of time.

All bottom times derived from the wheel will be more conservative than the theoretical model, but that is really not a problem, because the way it works out, air supply is

generally insufficient for the time limits allowed for most of the multi-level dives.

Should we stop at 15 feet for 5 minutes?

Even two years ago this issue was controversial in the U.S.A. Why would one want to stop? Well, obviously one experiences a large reduction in tissue pressure at a very critical point in the dive. If one started off at 100% of the theoretical maximum one could make a 3 minute stop and in the 5 minute compartment one would theoretically be down to about 80% of what one started with. If one made a five minute stop one drops to less than 70 % (Table 3). This outgassing is pretty significant. Obviously in a slow compartment one does not get much change. However, most people seem to think that neurological injuries occur through a faster compartment bubbling and in these large benefits are gained from stops.

TABLE 3

PERCENTAGE OF MAXIMUM TISSUE PRESSURE AT STARTING ASCENT AFTER STOPS

Half-time	Percentage of maximum pressure	
	After 3 minutes	After 5 minutes
5	78.9	69.1
10	89.8	84.1
20	95.7	93.1
40	98.4	97.3
60	99.1	99.0
120	99.7	99.5

The next benefit occurs if one stays too long or goes too deep. Going too deep or staying too long are essentially the same thing. Why would one either? I do not know. Sometimes it is equipment failure. It happened to me once, when my gauge was reading 85 feet and everyone else had 100. It did not matter then because the dive was short, but if it had been to the limit I might have been in trouble. If we use good diving practices and make this safety stop as well, it can cover many sins. It is the cheapest insurance policy that there is.

The main reason is to slow ascent rates. We know that people ascend faster than 60 feet a minute. It is almost a physical impossibility to stop at 15 feet if one is going up at 150 feet per minute. So we are already ahead of the game if one has people thinking about the stop.

Once one has stopped, one begins to accumulate the other benefits I mentioned. Possibly the most important factor, is that when one is ready to leave the stop and go on to the surface, in the few feet of the water column where the pressure gradient changes most dramatically as far as volume ratios are concerned, it would be very difficult to get going very fast in those last few feet. So we will bring them back alive.

And I think we will see a significant decrease in barotrauma, in lung expansion injuries, neurological effects,and that a lot of them would almost disappear if everybody started routinely making stops. After all, it only takes three minutes of one's time.

The American Academy of Underwater Science (AAUS) met for three days in September 1989[8] to argue about safe ascents. We reached consensus on about 20 different issues, but the most significant, the issue that the AAUS felt was the most important, important enough to put on the cover of the proceedings, was that divers stop in the range from 10 to 30 feet from three to five minutes. But the details are not the important thing. The important thing is the principle is not stop where one wants to, but stop somewhere near the surface.

The Diver's Alert Network (DAN) as recently as December 1989[9] issued a whole series of recommendations and guidelines for recreational diving in general. DAN recommended that ascent rates should be no more than 60 feet per minute and slower is acceptable. Stop at 15 feet for three minutes, or more, for all dives. We go along with that. A decal was issued more than two years ago, with PADI's "S.A.F.E." diving campaign, with the introduction of the slogan "Slowly Ascend From Every" dive.

Slow compartments exist, supposedly, down to extremely long halftimes of 480, 600 and 720 minutes. They are probably important in saturation diving, but not in the ordinary recreational experience. Nevertheless, there are rules printed on the Recreational Dive Planner which require long surface intervals on occasion. These rules exist to deal with slow compartments and we will discuss the point in the next session, but some published commentary has, at least mathematically, exaggerated the importance of these compartments.

Table 4 shows a list of the tissues on the left hand side. A list of depths down the middle, and a list of times down the right hand side. They are the time and depth combinations required to pump the tissues up to the maximum for the theoretical amount of time that the model confers. Now, I really do not think that most people are going to stay for approximately a day at 24 feet, or more than that at 23 feet, but that is what it takes. So, for single dives, these times are obviously irrelevant. But it can become relevant if one is doing a lot of repeated multi-day diving.

TABLE 4

DEPTHS AND TIMES WHICH CAUSE MAXIMUM TISSUE PRESSURE

Half-time tissue	Depth (feet)	Time (minutes)
80	32	281
100	30	360
120	28	487
160	26	723
200	25	929
240	24	1,265
360	23	1,892

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This is an edited transcript of a lecture given at the 1990 Annual Scientific Meeting of SPUMS.

A companion paper "Testing the Recreational Dive Planner" also edited from the lecture transcript will appear in a later issue.

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THE DITAA SURVEY
A REPORT ON A STUDY OF THE AUSTRALIAN DIVING INDUSTRY

Warrick McDonald

DITAA (Diving Industry Travel Association of Australia) is not just an organization to run SCUBA EXPO, the annual dive show where wholesalers, manufacturers and retailers exhibit their goods. DITAA takes its representation of the diving industry seriously and now offers members a wide spectrum of benefits. This survey is just one.

The survey was undertaken by Arthur Young and Company to benefit the diving industry through improved knowledge and understanding of the market and thereby help the industry along the road to greater success resulting in some cases in better profits.

Once one has analysed the contents of the survey one will appreciate just how valuable the information can be. If used wisely, the survey will enable the diving industry to plan and manage their businesses for profit. The information contained not only will benefit divers, dive shops, instructors, charter boats and wholesalers but other allied industries such as travel consultants, medical practitioners and printers of associated products.

The survey was commissioned by DITAA in order to provide, for the first time, information about the diving industry on a national basis. Little or no market research has been done for the diving industry in Australia. As a result it has been difficult for most operators to estimate the level and nature of demand for equipment and services. Retailers could lose in two ways if these demands are not known, either by having an inadequate supply of products from under calculation of current needs or by overstocking and possibly having to sell, at cut-throat prices, goods in an effort to reduce stocks.

The report of the survey analysed the current diving environment in Australia and discussed the study findings in detail. This paper will cover just a few of the points in the DITAA survey.

The Australian diving industry is affected by such things as population, external competition, customer lifestyles, technology, environment, overseas trends, the economy, internal competition and regulations. In 1981 the Australian population was 14.6 million, by the year 2001 it is estimated that the population will be 20 million. Also the age groups are changing. By 2001 28% will be 19 years old or younger while the 40-59 group will be 25%. From this the market will lie in the latter, "Baby Boomers", age groups and attention should be channeled into introducing them to diving. Customer lifestyles are towards family orientated,

less strenuous forms of exercise. Many new sports have been introduced to schools and competitive pressure is on businesses to upgrade their facilities and their approach to the market. Environmental issues are increasingly of concern to Australians.

Scuba diving is uniquely positioned to take advantage of the popularity of environmental issues. Technology has influenced the industry through new equipment and materials. Internal competition is very strong. Store ownership turnover is very high as divers do not necessarily make good businessmen. In the course of the study, the researchers found a degree of suspicion and mistrust amongst some members of the association.

Arthur Young and Company, now Ernst and Young, had some harsh words about professionalism in the industry "Although all the people contacted by the researchers were very friendly, there is undoubtedly a strong current of mistrust among the members of the industry which prevented a few of the major operators from participating. There is an apparent lack of professionalism which needs to be addressed frankly and openly as such attitude will ultimately be detrimental to the individual operator". Overseas the trend is toward customer-service orientation. This is the need to listen and cater to customer needs. The diving industry currently has in place a number of government and industry-enforced regulations regarding safety and the level of instruction.

Australia's economic future is uncertain to many of us, with high interest rates, lowering of the Australian dollar and a decline in the standard of living. The general prognosis is bad. Because the overall effect makes it more expensive to travel overseas, it may mean more dollars will be spent on local leisure activities. However, the initial costs of becoming involved in diving are becoming more expensive.

Response to the study was high in some fields but low in others. 75% of diving business operate from only one outlet and 56.6% are owned by companies. In 1988 49% of the turnover of the average dive shop was from equipment sales and 28% from their scuba school. Retailers' turnover was largest in Queensland then NSW followed by Victoria.

Certification agencies reported an average 16% increase in numbers certified. The largest group of students was aged between 19 and 35 with consistently more males than females. The average cost of an Open Water or Entry Level Course was $300, exclusive of any equipment hire or purchase which would be required to complete the course. To this must be added the medical and X-ray costs.

It is no wonder that diving instructors are becoming harder to find for the dive schools, as they only earn an average of $18,564.

It was shown that, consistent with world figures, PADI was Australia's largest training agency with a 51% market share. In 1988 50,550 students were trained as new divers. This figure would mean that approximately $122,330 was earned, on average, by each dive school from new divers. Some of these would purchase equipment and progress to continuing education courses, adding to the dive shop's income.

The Yellow Pages proved to be the next most popular form of advertising but had the lowest perceived effectiveness, whereas television, the least used medium, was considered the most effective. "Sportdiving in Australia and the South Pacific" was the most popular industry magazine. It was a shock to most people when it was revealed in the survey that the most popular dive travel destination was Queensland, not the Pacific Islands.

Many other facts and figures were gained through the study but they are far to numerous to mention here. The 32 page report of the study was sent to financial DITAA members and is on sale to non-members for $75.00 a copy.

Warrick McDonald is DITAA Secretary and a PADI Instructor and Course Director. His address is ab Ocean Divers, 237 East Boundary Road, East Bentleigh, Victoria 3165, Australia

DIVING TRENDS IN THE UNITED KINGDOM

Greg Adkisson

Accepting that man is a land, rather than an aquatic animal, man's excursions into the oceans, throughout history, have been limited more by technology than by human physiology. It is only within the last hundred and fifty years or so that technology has begun to outpace us. Today, we are faced with a new breed of diver armed with higher pressure bottles, warmer suits and an array of new equipment designed to allow deeper and longer diving while keeping decompression to a minimum. We know that sport diving groups are changing the way they dive and nowhere is this more evident than in the new BS-AC '88 decompression tables or in the new PADI wheel for multi-level diving. We must be aware of these trends and be knowledgeable in our efforts to express a considered, cautious alternative view.

Historical Analysis

To understand where we are going, it is often helpful to look back and see where we have been. There is an apparent increase in the number of diving accidents over the last few years but of more concern is that the pattern of illness may have changed as well. Studies throughout the years have divided decompression sickness (DCS) into non-serious, Type I (pain only) and serious, Type II (neurological/pulmonary) DCS. Historically, the percentage of serious DCS was stated at around 30%[1-3] with Erde and Edmonds' 1975 study[4] being a notable exception. Different population groups, data interpretation, what constitutes a Type II lesion and differing exposures all make comparisons of these studies difficult. There is clearly, however, a changing pattern of diving and it seems that the amount and pattern of DCS may be changing with it.

TABLE 1

PERCENTAGE OF CNS INVOLVEMENT IN DIVERS WITH DCS

1963	Rivera	26%
1965	Slark	35%
1969	Kidd and Eklliott	25%
1975	Erde and Edmonds	52%

From 1985 to 1989, the number of sports divers treated or advised by the Ministry of Defence, Navy (MOD(N)) has risen steadily. The most dramatic increase occurred in 1988 when there was a twofold increase in cases treated compared to previous years.[5] Central nervous system involvement among those divers treated for DCS also rose. It reached a peak in 1988 when 87% of divers treated showed evidence of CNS involvement. The figure was down to 76% in 1989, still a notable increase from past years. I was personally concerned that the divers I treated from '87 to '89 appeared to require more extensive therapy to achieve resolution than in my previous years of experience.

Factors Involved In Diving Casualties

What caused this increase is not entirely clear but a number of factors appear to play a role. Divers are going deeper and staying longer with the advent of new and improved equipment including warmer suits, larger air bottles and advanced technology in the form of diving computers and other diving aids. Sports divers regularly venture into depths that the commercial divers are restricted from entering. Decompression stop diving has become routine and new tables, designed to maximize in-water time, have been introduced into widespread use. In 1988, the use of diving computers was involved in 42% of the accidents treated by MOD(N) and the practice of repetitive diving in 63%. Depth in excess of 30 metres accounted for 68% of the accidents with 21% being greater than 40 metres and 12% from depths in excess of 50 metres.

TABLE 3

FACTORS INVOLVED IN 95 PATIENTS WITH DCS TREATED IN 1988

Dives deeper than 30 msw	68%
Dives deeper than 40 msw	21%
Dives deeper than 50 msw	12%
Repetitive diving	63%
Use of Computers	42%

Additional factors, perhaps more traditional, played a role in the 1988 figures. 54% of the divers treated in 1988 were older than 30 and 21% were over the age of 40. Diving at altitude accounted for 6% of the treatments while 5% were associated with flying after diving. Medical conditions which would normally render a diver unfit were associated in 10% of the cases. Whilst not involved in a recompression treatment, the oldest diver I was consulted about was 72 years of age and had just returned from a series of dives in an isolated area of the world without recompression treatment facilities. The oldest diver actually treated was 67 years old.

TABLE 2

SPORTS DIVERS TREATED OR ADVISED BY MOD(N)

	1985	1986	1987	1988	1989
Advice	71	69	82	143	145
Treatment	45	50	51	105	137
% of DCS Cases with CNS Involvement	51	65	68	87	76

Implications For the Future

No longer is diving a sport for a few hearty individuals. Sport diving has become big business with major commercial interests servicing all aspects of the sport; provision of gear, instruction of new divers and the arranging of diving holidays. Diving is expected to grow in popularity and, as the number of divers grow, it is likely that the numbers of diving related injuries will grow as well. Likewise, as the introduction of new and sometimes untested, or minimally tested, diving aids continues, the potential for more diving related injuries will increase. As many of our divers grow older, it is likely, as in the rest of medicine, that greater numbers of older patients will present, often with associated medical problems.

We have been concerned, as a medical community, that a great deal of long term neurological damage may be caused by diving, and that it is not always apparent by our existing test procedures. Whether this subclinical damage affects the patient later in life remains a matter of speculation. What does appear to be certain, is that, as technology advances and divers are urged to go deeper and stay longer, advancing medical technology will allow us to take a more critical look at the effects of this hostile underwater environment on our patients, both young and old.

Diving is a hazardous occupation and a hazardous sport. Few of us expect or desire the sport diving community to stop diving. We hope, however, that, as a community, they will recognize the current limitations of human physiology. When faced with the possibility of a few extra minutes on the bottom or the possibility of reducing decompression by a few minutes they will balance the benefit against the potential cost. That cost, all to often, is high. It may be a lifetime in a wheelchair, a life-altering disability or simply a nagging reminder of an old injury. Whatever the cost, major or minor, it is important for us to remind the diving community that the vast majority of so called "accidents" we see are, in fact, self induced injury that can be avoided with a reasonable degree of caution.

The implications for the working diver may be even greater. As techniques for examining the central nervous system become more sensitive, the question of short and long-term neurological damage will make an even greater impact on the commercial industries. New standards of medical qualification may become necessary. Baseline CNS studies, long term follow up and accident investigations may begin to document hitherto unappreciated neurological changes. If damage is documented, the question becomes whether the small degree of damage seen in some cases has any functional effect, either in the short or the long term health of an individual. These questions will undoubtedly be driven in part by medico-legal and occupational health considerations. The implications for the diving industry are enormous. As man's ability to dive ever deeper into the oceans develops, so does the appreciation of the dangers he faces. Now, more than ever, reasonable caution must be used whenever man, the land animal, enters the water.

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COURSES

UNDERWATER MEDICINE AND THE MANAGEMENT OF MEDICAL EMERGENCIES IN DIVING

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DIVING DEATHS DOWN AGAIN

Chris Allen

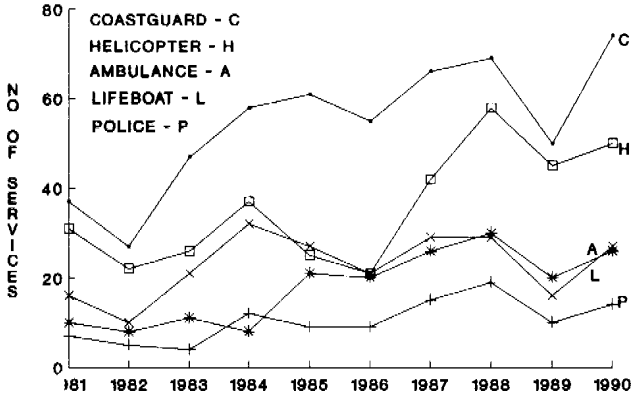
As the 1990 diving season draws to a close, it is once again time to look back over the year's incident reports and to reflect on the lessons to be learned.

Happily this year's safety performance has been particularly good and there are a number of encouraging trends. However, there are still lessons to be learned, some of which do not seem to change much from year to year.

As in previous years I think it is worth reviewing the background to the statistics first, before getting too deeply involved in their analysis. Those of you who read my report last year may remember that I felt that a number of changes in reporting procedures, together with a record breaking summer, had combined to influence the statistics.

This year we have again had a very good summer with generally good conditions for diving, particularly in the later months. However, from my own observations and the results of the surveys which we perform, I believe that the number of dives performed this year is somewhat lower than last year's figure.

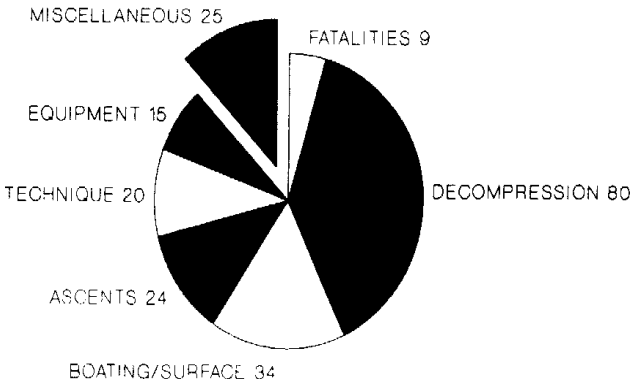
DIVERS' USE OF EMERGENCY SERVICES



As far as reporting goes, our link with the Coastguard is again working well, after a temporary lapse last year, and we have received substantially more information this year. The Diving Diseases Research Centre (DDRC) continue to supply us with brief details of the cases they have treated, as do other recompression facilities. We are indebted to them all for the valuable information they supply. Unfortunately our capture of data relating to decompression cases has slipped back a little this year, in that the Institute of Naval Medicine has not been able to supply us with all its data in time for inclusion in my published report.

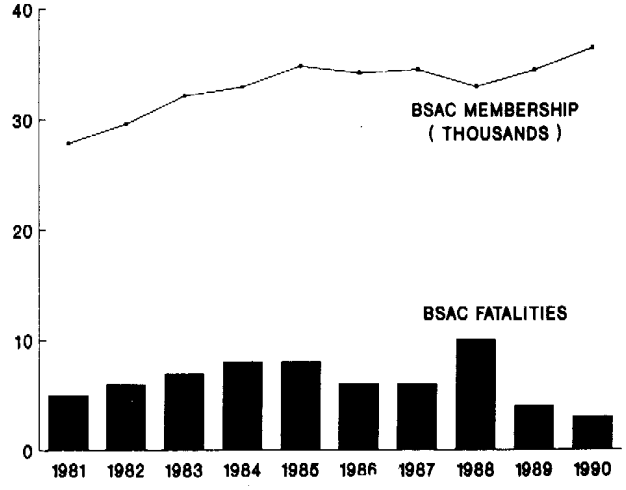
So with that background in mind, what do the 1990 statistics show ?

DIVING INCIDENTS BREAKDOWN – 1990



This year I am very pleased to be able to report a reduction in the number of fatalities for the second year running. A total of nine fatalities was recorded, including two deaths attributable to natural causes. Of those nine, just three were BS-AC members. This is the lowest number of BS-AC fatalities for thirteen years. Encouraging though this may be, every fatality is one too many and we must always aim to have none at all.

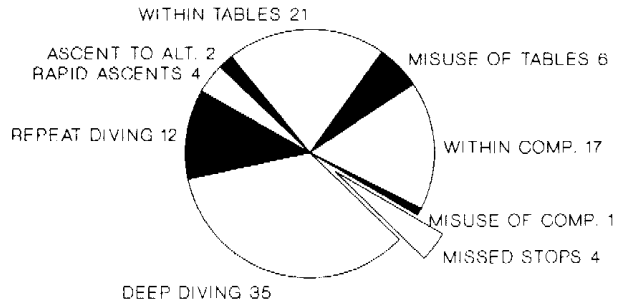
BSAC FATALITIES AND MEMBERSHIP 1981-1990



In recent years there has been a lot of publicity surrounding the number of cases of decompression sickness. It is therefore particularly pleasing to report a sharp drop in

the number of cases this year (from 137 to 80). Undoubtedly there had been some under-reporting this year because we have not received the full data from the Institute of Naval Medicine. However, I know from our regular contacts that they have seen a reduction in the number of cases they have treated, as have DDRC and other facilities. I am therefore confident that the total number of cases will turn out to be no more than 100. Even allowing for the fact that less diving may have taken place, this still represents a big improvement. I personally have no doubt that our efforts over the past three years to highlight the problems, together with the learning opportunities which the introduction of the BS-AC '88 Tables has presented, have also contributed to this lower figure.

DECOMPRESSION SICKNESS ANALYSIS – 1990



TOTAL NO. OF CASES = 80

Last year I highlighted the problems of divers running our of air, a factor involved in many recent fatalities. Although fewer serious incidents occurred in 1990, this is still an area of concern and there have been a number of near misses. In one case a pair of divers both ran out of air at exactly the same moment on a dive to 19 m. They both made rapid free ascents to the surface and one was later recompressed as a precaution.

In a second case a party of three divers went much deeper than originally planned. On the ascent one of the party ran out of air at 20 m and then made a successful assisted ascent to the surface. On the surface one diver had 2 bars left, one was completely out of air and the third still had 60 bars. All of them were using identical cylinders. This individual variability in the rate of air consumption and the dramatic changes which can be brought about by hard work, depth or cold, are precisely the reason why regular monitoring of both your own and your buddy's air supply is essential during every dive.

Proper planning is essential to ensure safe, adventurous diving and nowhere in the statistics does this become more evident than in the case of lost divers. Early this year a well organised group of divers were preparing to dive in a popular location when four divers surfaced close by, apparently without boat cover. Their boat turned out to be anchored unattended three quarters of a mile away. Having re-united the lost divers with their boat, the original party again prepared to dive. Whilst kitting up two more divers popped up close by, again without cover ! They turned out to be from a charter vessel also about three quarters of a mile away. These divers didn't even know the name of the vessel they were diving from. When they were nonetheless transferred back to it, it transpired that the skipper knew he had lost contact with the divers but was not endeavouring to search for them.

But for the presence of our diving good Samaritans, the lack of forethought and general lack of awareness by both sets of rescued divers could have been more serious.

This year a number of incidents have resulted purely because divers have ventured too deep. Two cases involved divers descending solo to great depths "because it was there". In one case a diver made a solo night dive to 84 m and in a second case a diver on holiday in the Red Sea just carried on down to over 80 m leaving his buddy behind. Interestingly both individuals were "found out" by the evidence of their dive computers and had the error of their ways pointed out to them in no uncertain terms by very responsible action from their respective branches. Such cases of sheer lunacy are extreme and fortunately rare. However a significant proportion of incidents occur in dives to 40 m or deeper, simply because the divers involved have underestimated the seriousness of their undertaking. At these depths the margins for error is dramatically reduced and such dives should only be carried out by experienced divers who are fit, well prepared and properly organised. In addition, just in case anyone should be in any doubt, it is perhaps worth re-stating that 50 m is the maximum depth for sports diving. Beyond this depth nitrogen narcosis becomes a very serious problem and the risk of decompression sickness is much greater. What is more, if you do get bent, the treatment also becomes much more difficult.

It is not for nothing that professional divers are restricted by law to dives of less than 50 m when using air.

This year there have been several cases of serious carbon monoxide poisoning recorded. Although, or perhaps because we emphasise the dangers in our training it is actually rare to find air which is dangerously contaminated. This year, however, a number of incidents have occurred, mostly centred around one particular compressor station.

In one case a diver became distressed and breathless underwater and had to be brought to the surface by her buddy. On the surface she was still weak and breathless and was flown to DDRC by helicopter for hyperbaric oxygen treatment.

That weekend two divers in the same area complained of being weak, dizzy and having disturbances of

vision. On the surface they were confused, uncoordinated and their speech was slurred. They too were flown to DDRC for hyperbaric oxygen treatment. Later tests found over 2,000 ppm of carbon monoxide in their cylinders (the BSAC recommended limit is 5 ppm).

In a third incident a diver became dizzy and completely disorientated on a dive to 15 m. Though he surfaced immediately he had only vague recollections of the ascent and being taken back to shore. Once again high levels of carbon monoxide were detected in his air.

It is perhaps fortunate that none of these divers was particularly deep as the higher partial pressures of carbon monoxide which would have been encountered could easily have caused sudden unconsciousness underwater. The lesson for us all is that dangerously contaminated air can still occur and that we therefore need to be vigilant. If contaminated air is suspected, do not take a chance and dive, for you could easily end up with something much more serious than a bad taste of headache. As a first aid measure in such cases, the administration of oxygen is helpful-one more reason for every diving party to carry an oxygen set !

As you can see from the preceding descriptions, the symptoms of carbon monoxide poisoning could easily be mistaken for those of serious decompression sickness. If faced with a diver exhibiting such symptoms, at least consider the possibility of a contaminated air supply. Incidentally, the rogue compressor responsible for most of the recorded cases was quickly shut down and investigation into the cause was launched.

Many of the incident reports I receive involve rescues performed by other divers.

One such case this year involved a group of army divers on an expedition to Norway. Just as they were finishing for the day they were alerted by a Norwegian diver whose buddy had been trapped underwater by a rock fall. A rescue operation was quickly launched with air being taken down to the diver who was trapped. The rock, which weighed more than 1,000 kg, was moved with the aid of lifting bags and the unconscious and badly injured diver was brought to the surface and given EAR until he was evacuated by rescue helicopter. Unfortunately the diver's injuries were so severe that he died later, but BS-AC Rescue Awards have been awarded to those involved for performing the rescue under such difficult conditions.

The potential hazards of falling overboard from a fast-moving boat were graphically demonstrated in a report concerning a diver who entered the water by performing a backward roll entry from a moving rigid hull inflatable. He sustained a fractured skull when he struck his head on his pillar valve, and at the same time burst one of his eardrums due to the sudden water pressure.

In diving a small incident, which on its own would not be a problem, can become life threatening when other things also start to go wrong at the same time. Once you start to slip into the "Incident Pit" it becomes more and more difficult to escape.

A keen novice diver from one of our overseas branches was preparing to dive from a hard boat. She had a little too much weight on and had not realised that the cylinder she was using was heavier than the one she normally used. As she was getting ready she slipped and fell and her cylinder slipped out of her stab jacket. She was helped up and her bottle and jacket were reassembled. During this time it appeared that her air was turned off.

Having sorted herself out she decided that since it was so hot and the boat was crowded, she would enter the water to cool down, fit her fins and do a buddy check. When she jumped in she sank straight to the bottom with her air turned off and no fins on !

She was underwater for several minutes before another member of the party was able to dive down and bring her to the surface where she was successfully resuscitated.

Another case which happily also ended safely, though it might so easily not have done, involved a a diver who had made a dive to 60 m in a quarry. Just as he began to ascend his regulator began to freeflow. He decided (at 55 m!) to take his set off and switch it off, changing over to his pony cylinder and spare valve at the same time. Unfortunately he got in a tangle with his equipment, his mask flooded and he began taking in water. Somehow he reached 25 m where he managed to sort himself out, at which point his pony cylinder ran out. He managed to turn his main set back on, replace his equipment and, after performing the required decompression stops, surfaced a much wiser man !

Those, very briefly, are some of the incidents which figure in the 1990 Incidents Report. I would urge everyone to ask themselves whether some of the incidents reported could have happened to them or their branch. If so, spend some time thinking about what needs to be done to make sure they can't happen.

Our sport is actually a very safe one and overall it has been a good year. However, we must never become complacent. The best way to avoid featuring in the 1990 statistics is to plan thoroughly, watch your depth and, above all, be aware so that you can anticipate problems and deal with them while they are still small.

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WHY U.S. DIVERS DIED IN 1986 AND 1987

The National Underwater Accident Data Center (NUADC) at the University of Rhode Island investigates and analyses the diving fatalities of United States citizens wherever they were diving. John McAniff, the director the Center, has been involved in the collection and analysis of these data for more than twenty years.

The National Oceanographic and Atmospheric Administration (NOAA), the Diving Equipment Manufacturers' Association (DEMA) and the Professional Association of Diving Instructors (PADI) are the primary funding sources. Newspaper articles provide the initial lead on two-thirds of the fatalities. The remainder come from contacts throughout the diving community and public agencies.

For several years, *Undercurrent* has been analysing these reports, editing and condensing them, and sharing the relevant data with our readers. We believe that by reporting the unique and varied circumstances in which divers die, we all may learn how to become safer as we conduct our own dives.

For 1986 NUADC investigated 116 diving fatalities, including 94 sport diver fatalities, 11 occupational fatalities and 11 skin diving (breath-hold) fatalities. For 1987, 108 cases were investigated, including 87 non-occupational, 12 occupational and 9 skin diving fatalities.

Last year DEMA commissioned a study by Diagnostic Research, Inc. (DRI), which estimates active diver population 2.1 and 2.7 million. Based on these figures, the fatality rate for 1986 is 3.65 to 4.5 per 100,000 active divers. The computation for 1987 is 3.4 to 4.2 fatalities per 100,000 divers.

The population of active divers in the United States has been steadily increasing, while the number of fatalities has fallen off considerably. In 1976, for example, we recorded 147 sport diving fatalities, which equated to a 8.62 per hundred thousand fatality rate at a time when the population of active divers was probably one-third less than it is today.

Deaths by Locale

For 1986 and 1987, Florida recorded 49 diving fatalities, including nine cave diving deaths. Non-cave diving fatalities in Florida have increased considerably. California recorded 33 fatalities in the two-year period; Washington State 14; Hawaii 9; New Jersey 7; Idaho 6; New York 6; Texas 6; Massachusetts 5; Arizona 3; Tennessee 3; Connecticut 2; Pennsylvania 2; Puerto Rico 2; The Virgin Islands 2.

The incidence of US sport diver fatalities in foreign areas has been highest in Mexico, which since 1970 has totalled 37 such fatalities. Eight deaths were recorded for 1986-1987.

NUADC has received an increasing number of complaints of diving conditions in Mexican waters, especially in Cancun and Cozumel. Among these complaints have been reports of introduction of novice (short resort course) divers to swift currents and deep reefs on early dives, lack of adequate supervision, lack of knowledge or facilities for the treatment of injured divers and absence or lack of ship to shore communications.

Other foreign areas reporting deaths of US divers during the two years include the Bahamas with 3 deaths; Canada 2; the Cayman Islands 2; Belize 2; Honduras 1; and Saipan 1.

Environmental Aspects of Fatalities

Approximately 66 percent of all sport diving fatalities occur in salt water. Diving in fresh water caves continues to be a source of problems for sport divers; 18 fatalities occurred over the two-year period.

Each year, the NUADC receives numerous requests from individual divers for information to help them in their attempts to obtain life insurance. For some unobvious reason, many insurance companies tend to rate divers on whether they normally exceed an arbitrary depth of 50 feet in their diving. The NUADC has found no evidence to support the 50-foot depth as a demarcation point for rating of life insurance for sport divers. Life insurance can be had for varying prices by shopping around to different insurance companies to locate those that do not use the rating system for scuba divers.

Environmental or weather conditions have contributed to diving fatalities in surprisingly few cases over the years, and these are usually the fault of the diver for not having investigated conditions before entering the water. This is especially true for strangers visiting a new area. An example might be Florida divers who go on vacation in California or Hawaii and suddenly finds themselves faced with heavy surf with which they are unfamiliar.

A considerable number of sport diving fatalities have occurred from charter boats, most likely a function of the tremendous growth of the charter business. There is a need to establish standards of safety for such vessels.

Age and sex distribution

In our last report*, the NUADC noted an increase in fatalities of individuals over 50 years of age. This apparent trend has continued for both 1986 and 1987; 18 percent of the fatalities were over 50 years of age. It is impossible to determine whether this trend reflects an older age group entering the sport or older divers who are diving into their later years.

But the young are not excepted. In 1986, a 12-year-old boy died while diving in 55 feet of water from his father's private boat. In 1987, a 10-year-old boy died in formal training in open water.

Seventeen percent of the fatalities were female.

Starting causes of deaths

The starting cause for some cases is impossible to determine. Nonetheless, the NUADC considered 30 cases stemming from possible exhaustion, embolism or panic.

We often hear that panic causes a large number of diving fatalities. It might be better expressed that panic is a stepping stone to eventual air embolism or drowning. Some other events must be present to start the panic cycle: the flooding of a mask, the loosening of a weight belt, a cut on one's finger, the unexpected exhaustion resulting from swimming against current, a sudden storm surge. Hundreds of other possibilities may be the starting factors in a fatality.

A typical scenario might be as follows:

A 19-year-old boy, who has recently completed an open water diver certification and has made two or three dives with a friend, books passage on a charter vessel to delve the ruins of a fishing vessel in 60 feet of water. The weather is overcast and there is a steady roll to the water, adding to the queasiness of the boy's stomach. On the way to the wreck site, the divemaster chooses a buddy for the boy, a 34-year-old female who has a considerable amount of diving in her expertise.

Upon anchoring, the boy is quiet and sullen while the woman is enthusiastic and anxious to enter the water. The visibility is only about eight feet. As a precaution, the two proceed on their dive holding hands as they submerge. The usual difficulty with clearing the ears takes a few seconds. By the time they have reached the bottom, the boy's sullenness has disappeared and he has become fascinated by the wreckage. The buddy diver, feeling confident in the boy's new-shown spirit, moves some distance away to the edge of his vision. The boy momentarily panics and moves swiftly after her, at which point he feels he is having difficulty breathing.

His signal to the buddy results in an attempt to place her mouthpiece in his mouth. Instead, his mask is knocked loose, he bites down hard on his own mouthpiece and begins to choke a little on water. On the edge of panic, he reaches to release his weight belt, instead opens the buckle for his dive tank harness. The tank assembly begins to drift away from his back and he begins flailing with both arms and legs while fighting to reach the surface. Panic has now set in. The buddy diver backs off and loses sight of the potential victim. She surfaces, only to find no sign of the boy. Four other divers enter the water, swim to the bottom and find him sitting there with his face looking upwards, no mask in place and the regulator out of his mouth, a classic panic/embolism case.

We have 13 deaths in 1986 and 9 fatalities in 1987 that could not be identified as having had a different probable starting cause than the embolism itself.

A 23-year-old male snorkeller dived down to 25 feet to two friends who were scuba diving. He stayed there several minutes, borrowing air with his friends. Upon leaving the friends on the bottom and coming to the surface, he popped right up out of the water. He threw off his mask and snorkel and went limp. He had embolised, a result of breathing compressed air then surfacing without exhaling, appropriate only if his initial breath of air had been at the surface.

Alcoholic intoxication was cited as the probable starting cause in at least four cases in the two years of study.

A 34-year-old man died in a marina in San Diego Bay when he intended to work on his boat. He stepped off the swim step with his fins in his hands and mask on his forehead. He was reported to have been wearing 16 pounds of weight and did not have the regulator in his mouth. In less than ten minutes, the body was recovered in 26 feet of water directly beneath the boat. The autopsy found that the deceased's blood alcohol level was 0.23.

Cocaine contributed to at least three cases over the two-year period. Two deaths were attributable to nitrogen narcosis; in one of these cocaine was a contributing factor.

In separate cases, the boat, left empty but anchored, drifted away from the divers. One diver died in each incident.

One death in Hawaii occurred when a diver suffered severe lacerations and fractures of the head and torso from the propeller of a passing boat.

The NUADC found several cases in each of the two

* *See Lippmann J. How do American Divers Die? SPUMS J 1988; 18 (3): 104-106.*

years in which the victim was overweighted, though this may not be considered the starting cause of the accident.

Equipment failure is seldom the cause of a diving fatality. The glaring exception is the improper or poor maintenance of a diver's regulator.

Experience and training

Early entrants into the sport accounted for 23% of the casualties during the two years. Yet, the great majority of fatalities during these years were individuals whose experience range from "some" to "substantial".

In 1987, there were seven deaths of students in beginning or advanced training and in 1988 there were eight deaths. Some of the cases from which we might learn lessons include:

A 64-year-old male who had been diving for 18 months and had completed a basic and intermediate training course was undergoing an advanced diver course. He and his buddy had been operating at 110 feet when the victim suddenly rushed for the surface. Upon surfacing, he appeared to be alright until he reached the boat, when he went into unconsciousness. CPR was attempted but it was impossible to revive him. The death was due to an air embolism.

Off the coast of Monterey, California, a 55-year-old male was in an advanced diving class. He had made an uneventful dive to 80 feet for 20 minutes with a normal ascent, but soon after surfacing he became unresponsive then went unconscious. Extensive CPR was immediately started, but was unsuccessful. The autopsy indicated asphyxiation due to drowning with no indication of any embolism or coronary condition.

Diving partners and their activities

For 1986 we recorded five incidents in which two persons died and one case in which three persons lost their lives. The 1987 statistics show two double fatality cases and one case of three deaths in the same incident. Most of the multiple fatality cases over the years of this study have involved diving in caves.

The double fatality of a pair of young men, 28 and 33 years of age, that occurred on a beach on Long Island Sound apparently went unwitnessed. Neither of the two divers had attempted to ditch his weight belt and both died of drowning.

An engaged couple died off the coast of Cozumel. The 23-year-old male, who had only limited experience in scuba diving, suffered an air embolism. The body of his 23-year-old fiancee has never been recovered.

In several fatal incidents, three or more people were diving together. NUADC believes that a group of three or more in the water for diving purposes immediately destroys the concept of the buddy system. It is often impossible to determine who is acting as a buddy to whom, and in the event of an emergency, leads to utter confusion. We continue to emphasize that a buddy system consists of a one-on-one buddy pair, each capable of looking out for him or herself and at the same time being close enough to be of assistance to his buddy should the need arise.

Deaths during instruction

Several training fatalities involved older persons. These students require a more stringent physical examination to determine whether they can withstand the extra stress and effort of diving.

An embolism was the cause of death of a 27-year-old female who had been conducting a free swimming ascent with her instructor from a depth of 30 feet of water in a fresh water facility in New Mexico.

A 40-year-old woman who had displayed classic symptoms of apprehension during her pool training was about to take her first open water dive in the Gulf of Mexico. Almost immediately after entering the water, she felt so nervous and uneasy that she grabbed the anchor line of the dive boat. A wave swept her against the boat, but not hard enough to knock her unconscious. However, she suddenly collapsed. Despite all efforts at CPR, she died in the water. The doctor who conducted the autopsy was quoted as saying that she probably developed a very rapid heartbeat when experiencing extreme nervousness, then apparently went into cardiac arrest.

An instructor must be constantly looking for the signs of anxiety, nervousness or fear on the part of a student. This is especially true of the first open water experience. Several of the cases cited were the result of such anxiety with panic and air embolism resulting.

An Oregon boy who reached his 10th birthday in March of 1987 died less than three months later on a beach in Seattle while engaged in an open water scuba class in which his father was also a student. The instructor and seven students, including the victim, had made a successful swim underwater to a buoy approximately 100 yards offshore. All of the students were returning, with the victim next to last on the underwater line with his father following him. Visibility was less than 18 inches. The boy surfaced without his mask on and his regulator out of his mouth, yelling for help, then

disappeared below the surface. The boy was not missed until the father and other students arrived on shore. The victim's body was recovered after a two-hour search. Apparently the boy was wearing a wet suit too large for him with the sleeves and cuffs rolled up; he wore approximately 25 pounds of weight.

The death of a 42-year-old male took place on a Cape Cod beach during the victim's first open water exercise. The instructor entered the water with six students on a swim to a marker buoy and dive flag about 100 yards off the beach. The instructor proceeded to the bottom with four of his students while leaving the victim's buddy at the buoy to wait for the victim to reach the buoy. Winds of 20 to 25 miles per hour with 4- to 5-foot swells may have affected the situation. The victim was wearing about 36 pounds of lead, considerably more than necessary for his wet suit and other equipment. The victim called for help once or twice and was almost immediately pulled ashore. However, he was unconscious and did not respond to extensive resuscitation efforts.

Nearly every year of this study we find early open water fatalities resulting from the improper weighting of a student. Special care should be taken by the instructor on every first open water dive to determine proper buoyancy for each and every student.

At Jamestown, Rhode Island, a dive plan was laid out by an instructor for a simulated decompression dive of 90 feet for 20 minutes. During this exercise, the 35-year-old male victim indicated to his buddy after 17 minutes of diving that he was out of air. The two apparently buddy breathed up to 50 feet, where the buddy thought that he too was out of air. He tied off the victim at that depth and raced for the surface and called for help. An immediate search located the victim at a depth of about 50 feet, entangled in line. Extensive CPR efforts were unsuccessful and the autopsy revealed death from embolism.

A freshwater-filled quarry in Pennsylvania was the site of the death of a 22-year-old female who was engaged in her second checkout dive during instruction. This victim apparently drifted away from her buddy diver and was not found until an hour later in 80 feet of water. The diagnosis was embolism.

A state beach in Los Angeles was the location of the death of a 19-year-old male who was undergoing a final checkout dive for basic certification. He had been accompanied by another student and an assistant instructor. Upon completion of an exercise, the victim was escorted to the surface and told to wait there until the other students had finished their exercise. Upon surfacing the victim was missing. He was soon found 30 feet deep, on the bottom with his regulator out of his mouth, mask off and one fin missing. Despite extensive CPR efforts, he was pronounced dead due to drowning.

It seems imperative that students be under close supervision at all times during open water training. Leaving an individual student with an equally inexperienced buddy is an invitation, not only to a single disaster, but also to a possible double fatality.

Cave diving

Cave diving accounted for 10 deaths in 1986 and an additional 8 deaths in 1987, most of which occurred in Florida caves. Between 1960 and 1987, 261 divers have succumbed to the lure of Florida's freshwater caves.

Nearly all of these cases have been open water divers who have had little or not training in cave diving, who entered a cavern situation without the proper diving gear, violated the need for more than one light per diver, failed to lay an exit line for safety, and violated the rule of returning from a penetration after one third of one's air is used. All but one of the 1986-1987 deaths were classic examples of this.

A female diver had just been certified as an open water diver earlier on this fateful day. Her 37-year-old male instructor, the student, and two others attempted a cavern dive after dark without any proper cave diving equipment. After a short period in the cavern, two of the divers indicated they were low on air and surfaced. The instructor and the student indicated that they would stay a little longer. It was a dark night with no moon and the two victims were apparently unable to find the exit from the cavern. Recovery divers found both bodies at a depth of 80 feet, without their lights on and only dim illumination from two cyalume sticks. The student died of severe embolism. The instructor, too, did not make it out. He drowned.

There is no excuse for the tragic double deaths of an instructor and student in a cave or cavern situation. All instructors should be aware of the great danger to open water divers who are foolish enough to enter a cave or cavern situation without extensive training and proper equipment. Statistics do not lie. Diving in caves kills open water divers.

Another senseless death in 1986 on Easter Saturday at Peacock Springs. This accident was extremely well documented by members of the National Speleological Society-Cave Diving Section, who observed the start of the tragic dive.

The fatal group consisted of three men and a woman from Augusta, Georgia, all well equipped for a pleasant ocean dive with gloves, snorkels, single dive lights and no guide lines. The veteran cave divers on site attempted to explain the potential dangers of this cave system for penetration by open water divers. The group apparently listened, but paid little attention and proceeded with their dive. The veteran divers' last words to the group were "stay in sight of

the opening; we do not wish to make a body recovery so early this year".

About 30 minutes later, the woman diver surfaced and, upon questioning by one of the veterans, was unable to give any information, but the worst was expected. Two other members of the group then surfaced and confirmed that a diver was missing in the caves. The veteran divers attempted to rescue the missing diver, but after 20 minutes of searching, it became evident that they were dealing with a body recovery. One of the veteran divers reported:

"We located the body of the victim, a 31-year-old male, at the end of a gap line. He was pointing out of the cave, his single light was still burning, his regulator was out of his mouth, he was lying on the floor of the cave and there was no air left in the single 80 cu.ft. diving cylinder. The victim was less than 75 feet from an exit to the cavern and approximately 150 feet into the cave system. The body was located less than 45 minutes after the search began."

"None of the four divers in the victim's group was trained in cavern or cave diving. None thought to utilise a continuous guideline to the surface nor to allow at least two-thirds of the starting air supply for their exit. Each member of the team used only one dive light. The victim had been certified as an open water diver in May of 1985. He had recorded 37 open water dives."

A double fatality occurred in Morrison Springs near Ponce de Leon, Florida. Two men, both aged 25, planned a night dive into the spring. Both were using single tanks and were each equipped with a single rented underwater flashlight. Both divers were open water certified but relatively inexperienced. Neither had any cavern or cave dive training. Visibility in the spring was very limited. No guidelines were used, there was no evidence of use of the 1/3 rule for air, there was no backup air or backup lighting. It was alleged that some alcoholic beverage had been consumed before the fatal dive. The lack of experience, lack of training, and certainly the violation of virtually all cave diving rules took the lives of these two men.

In a particularly tragic incident, three brothers from the state of Iowa, aged 23, 29 and 31, lost their lives in the Devil's Eye cave system on the Santa Fe River in Gilchrist County, Florida. They left behind two widows and six young children.

A 28-year-old male died at Bonnet Springs near Live Oak, Florida. The victim and his wife had entered the spring and very quickly stirred up silt. The wife became frightened and left immediately. The victim, however, continued further on into the cave. Recovery divers stated, "A completely typical textbook-type open water diver cave diving with all the rules broken".

The death of a 17-year-old boy in a cave near Weeki Wachi Springs, Florida, created a veritable flood of newspaper publicity. This victim had entered the spring despite signs that were posted reading "No Diving". He had 100 open water dives and planned to take a cavern diving course, but could not wait before challenging cave diving. Apparently realising that he was lost almost 60 feet down and about 160 feet into the cave, this boy wrote a last message onto his tank with his diving knife. The message read, "I love you Mom, Dad and Christian".

One headline following a double fatality in the popular Peacock Springs area read, "Pair who knew better drown during cave dive". Both the 34-year-old female and the 32-year-old male were experienced open water divers. The latter, in fact, was to have been certified as an instructor on the day of his death. The two had apparently penetrated the cave for a distance of 150 feet and to a depth of 70 feet when they ran out of air.

For the first time in several years, the NUADC must report the death of a diver in a cave who apparently did have some training. This 25-year-old man had just recently completed a cavern diver course. He was attempting a penetration that even the most experienced cave divers would not try. The victim dropped a concrete anchor 20 or so feet down into a cave shaft against a very fierce upward current and then attempted to pull himself further into this penetration. He apparently lost hold of the rope and was forced backward against an overhead rock. At the same time his mask was blown off and his regulator was ripped from his mouth. Since this incident occurred at a depth of 120 feet, the recovery divers had to exert strong physical effort and overcome the effects of nitrogen narcosis to cut the equipment straps away from the victim's body and extract it from this deep hole. The expert cave dive investigator who recovered the body said: "This victim attempted to accomplish something that a reasonable and prudent diver would never have considered".

Though they certainly get much of the headlines, the beautiful fresh water spring and cave system of Florida is not the only area in which diving in caves results in fatalities.

During 1986, the NUADC recorded the death of a 23-year-old man in a cave in the ocean off Cancun, Mexico. A double fatality occurred in an underwater cave system off the coast of Belize, Central America. An American diver reported to have dive master qualifications, together with a Belizean who was reported to be one of their most experienced scuba divers, both lost their lives when they became lost in the cave system.

In July, 1987, an area called Shark Cove, north of Honolulu on the island of Oahu, was the location of a triple cave diving fatality. Three young US Marines in their early

20s died while exploring the salt water caves that are the result of lava flows. After they entered the caves, a strong tide and swirling water stirred up sand and destroyed visibility, making it difficult to find the way out of the caves. The victims did not carry lights and did not have any kind of a guideline back to the exit of the cave.

Wreck Diving

Five deaths each were recorded for 1986 and 1987 while the participants were engaged in diving on submerged wrecks.

On a submerged wreck off Key West, Florida, a 35-year-old female had completed a dive with her husband and another partner to a depth of 90 feet for 14 minutes. As she followed the anchor chain up to the surface, she suddenly started sinking back toward the ocean floor. The victim was quickly recovered and brought to the boat, where extensive efforts at CPR were not successful. She died of an air embolism.

All factors in this case point toward a too-rapid ascent. Many sport divers fail to adhere to the standard rate of ascent of 60 feet per minute or one foot per second (it is often recommended that you rise no faster than your smallest air bubbles).

A 25-year-old man lost his life while executing a six-minute decompression dive to 190 feet, beyond the limits recommended for sport diving. Upon the return trip to the surface, the victim stopped at 160 feet and indicated that he wanted to work on a porthole. His buddy proceeded upwards to continue his proper decompression stops. After 40 minutes under the water, the buddy surfaced and reported the victim missing. The recovery of the victim was hampered by several events. The second recovery team down sent the victim's body to the surface with a lift bag, only to have it drop back to the bottom. The ship's anchor began to drag and had to be reset. The victim was not recovered until the next day. In the meantime, fleshy portions of exposed skin had been eaten by marine animals. Death was due to air embolism with the use of cocaine possibly a contributing factor.

Two Americans, a mother aged 46 and her son aged 17, died while diving on the wreck of the "Arabia" at the Fathom Five Provincial Park, Tobermory, Canada. The two victims were diving as a group of three with the boy's father. Toward the end of the dive, the mother's tank had come out of the backpack harness and she and her son tried to hold it in place while rising to the surface, only to find that they had sunk back to the bottom. The two victims were later found by police divers and an autopsy cause of death in both cases was listed as massive air embolism.

Off the coast of New Jersey, a 34-year-old man died after becoming separated from his partner and snagged amid wreckage of a ship on the bottom of the sea. Autopsy conclusions indicated that this diver expired due to an air embolism.

An air embolism was also diagnosed as the cause of death of a 46-year-old male diver who was wreck diving 25 miles south of Beaufort, North Carolina. This death occurred after the victim had attempted to buddy breathe to the surface. He appeared to be alright until just before reaching the boat, when he stopped breathing and became unconscious. Extensive resuscitation efforts were undertaken, but the victim was pronounced dead on arrival after a helicopter flight to the hospital.

A 39-year-old man became entangled in cables while diving at a wreck in 120 feet of water 30 miles off Ocean City, Maryland. He and the buddy diver were rushed by helicopter to the University of Maryland Medical Center decompression facility, where the victim was pronounced dead. The buddy diver survived after recompression treatment.

A 120-foot dive to a wreck of a sunken steamboat in the St. Lawrence River took the life of a 33-year-old man. The victim apparently became disoriented, wrestled with his partner and then proceeded to dive deeper into the ship. The victim had been at a depth of 110 feet for about 20 minutes when this incident occurred. His body was not recovered until the following afternoon and the cause of death was listed as asphyxia due to drowning.

Ice Diving

On the Ipswich River in Essex, Massachusetts, a volunteer recovery diver drowned while conducting a search for a child who had fallen through the ice. Apparently, in the confusion of the scene, the diver was not known as missing until the following day. He was found under the ice on the opposite side of the river from the drowning site of the child. There is no apparent explanation for how such a diver could be lost except that he was a private volunteer of whom no one kept track.

In January of 1987 the owner of a dive shop in Montana was attempting to salvage a truck from 40 feet of water in a local lake. While attaching a tow cable to the truck, the diver apparently let loose of his safety line to the surface. A tender nearby noticed bubbles coming up under the ice and saw the victim had expired beneath the ice.

Medical aspects of fatalities

During 1986, the NUADC received autopsy reports on 125 cases. In many instances the body of the victim is not

recovered and some political jurisdictions have strict laws against the release of autopsy material, even for scientific purposes.

"Asphyxiation due to drowning" was the recorded cause of death in 41 of the 1986 sport diver fatalities and in 39 of the 1987 cases. Certain prosecutors are unaware of the problems of hypobaric (sic) that enter into a diving fatality case, and perhaps as many as one-quarter of those deaths that are listed as drowning may in fact be the result of barotrauma and embolism.

Twenty-nine were diagnosed as embolism. Some fatalities seem to start when the relatively inexperienced diver is confronted by a disturbing problem that leads to panic and a rapid escape to the surface. Several cases each year occur during the training for free swimming ascent. And there is a tendency of sport divers to exceed the mandatory rate of ascent of one foot per second.

Cardiovascular problems were diagnosed at autopsy in six of the 1986 cases and eight of the 1987 fatalities.

A 36-year-old mother was diving with her husband and their 11-year-old daughter in 15 feet of water off the coast of Washington state when she surfaced to complain that she thought she was having a heart attack. She had difficulty breathing, lapsed into unconsciousness and was pronounced dead at the hospital despite heroic resuscitation efforts. The autopsy finding was "probably cardiac arrhythmia due to over-exertion while scuba diving". (This is a cases believed by the NUADC to have been a possible arterial gas embolism and may have been discovered if the proper autopsy protocol had been followed.)

In approximately the same location as the previous case and two weeks later, a 48-year-old male succumbed and was diagnosed as having had coronary artery atherosclerosis and myocardial hypertrophy. This victim was also said to have had a therapeutic level of ephedrine (a bronchodilator, available in both prescription and non-prescription drugs) detected in the blood, which may have been a contributory factor to susceptibility of cardiac arrhythmia.

A 53-year-old male diver was found floating face-down off a beach in Hawaii. This man had a history of circulatory problems and was diagnosed as having died as the result of myocardial necrosis. He was diving alone.

A history of hypertensive cardiovascular disease preceded death of a 55-year-old man who had been diving with an excursion group in the Virgin Islands. The man was apparently an experienced diver who suffered from slight obesity and an autopsy determined that generalised arterial sclerosis was present.

Following a dive on the day previous to the fatal event, the 65-year-old male diver complained of a tightness in the chest. On the day of his death, he made two dives of 30 feet for 30 minutes and complained of being extremely fatigued. He collapsed in the water and was found floating face-down. The autopsy result indicated cardiac arrest complicated by occluded coronary atherosclerosis. The victim was six feet tall and weighed 235 pounds (107 kg or 16 stone 11 lb).

A 47-year-old male exhibited extreme panic and apprehension while diving in the Florida Keys. Upon re-entering the boat, he collapsed and was later diagnosed to have died of myocardial infarction.

Physical exertion while assisting in an advanced diver course was said to have contributed to the death of a 39-year-old male in Long Island Sound. Death in this instance was attributed to a heart attack.

The Atlantic Ocean off the east coast of Florida was the site of a fatal dive by a 39-year-old male who was reported to have been in good health and extremely active physically. He was found in 75 feet of water. Autopsy results indicated he died of over-exertion leading to a heart attack.

Less than one-half of one percent of all victims under the age of 35 show the cause of death as being cardiac-related; however, in those victims over the age of 35, 22 percent have been cardiac-related deaths. The NUADC is also noticing a larger percentage of fatalities amongst those in the older age groups. Most of these observations indicate a need for more careful screening and perhaps more intensive physical examinations for those in the older age group who are entering scuba diving.

The health and safety of incoming participants to the sport of scuba diving must be held uppermost in the minds of instructors and their parent training agencies.

Conclusion

The more we understand the tragic errors of our fellow divers, the safer we can make our own diving. A vast majority of the deaths we reported were unnecessary: the diver made a fatal error. Reporting these errors, we trust, will help you not repeat them.

Reprinted, with minor editing, by kind permission of the Editor, from UNDERCURRENT, 1989; 14 (7):10-12, 14 (8): 10-11, 14 (10): 9-11, 14(11&12): 11-12, 1990; 15 (1): 10-11 and 15 (2): 10-12.

The address of UNDERCURRENT is P.O. Box 1658, Sausalito, California 94965, USA.

DIVING INCIDENT REPORT FORM

Dr Chris Acott has provided a copy of the current diving incident report used by the Diving Incident Monitoring Survey (DIMS), run by the Royal Adelaide Hospital Hyperbaric Medicine Unit, for publication in the Journal.

DIVING INCIDENT REPORT

On average, one or two divers die each week in Australasia. Often "near-misses" can help to discover information to find a reason for these deaths. Incident reporting is part of this process. It has been used with great success in aviation and medicine. We believe that details of incidents lead to improvement in equipment and training.

Filling out this questionnaire may at times prove tiresome, but we urge you to do it as soon as practicable following the dive. If you participate you will assist in improving the safety of diving for everyone. Thank you.

Don't waste your valuable experiences - share it for the benefit of us all.

This study will form part of - indeed an extension of - the existing data that exists in Australia and New Zealand for the study of diving safety (presently known as "Project Stickybeak").

Definition of a Diving Incident

Is any error that could, or indeed did, reduce the safety margin for a diver on a particular dive. The error may have been made by a diver, either yourself, your buddy or someone else. It may also be due to equipment failure. Most incidents don't cause any harm, but reporting such incidents will give valuable information when considered with other such incidents.

Please **DO NOT** identify any person involved.

Dr. Chris Acott from the Hyperbaric Medicine Unit at the Royal Adelaide Hospital will be coordinating these reports - feel free to discuss with him any incident or any difficulties which you may encounter with this form.

If you would like any feedback from this incident, please include your name and address on a separate piece of paper and return it with this report form

Please return the completed form to:

APSF/DIVESAFE
G.P.O. Box 400,
ADELAIDE SA 5001 AUSTRALIA
(08) 224.5544

1

DESCRIPTION OF INCIDENT

Please describe the incident in your own words. Include in detail any factor which you believe may have contributed to, or minimized, the incident.

Suggest any measures which you feel might be employed in the future to prevent such an incident happening again.

2

THE INCIDENT ITSELF

ALL THE FOLLOWING RELATE TO THE DIVE INVOLVED IN THE INCIDENT, HOWEVER THEY DON'T HAVE TO BE FILLED OUT BY THAT DIVER INVOLVED, BUT BY THE PERSON NOTICING THE INCIDENT.

1. Whose incident was it?

   ☐ yours ☐ your buddy ☐ someone else's

2. When was it detected?

   ☐ preparation ☐ during dive ☐ exit
   ☐ entry ☐ ascent ☐ following exit
   ☐ descent

3. Did any harm result to anyone? Yes ☐ No ☐

4. Do you think any of the following factors contributed to the incident (you may need to tick more than 1):

   ☐ anxiety about the dive
   ☐ inexperience in diving
   ☐ weather conditions
   ☐ poor physical fitness
   ☐ failure to check equipment
   ☐ haste
   ☐ inattention
   ☐ poor dive planning
   ☐ poor servicing of equipment
   ☐ sea sickness
   ☐ insufficient training
   ☐ not familiar with diving conditions
   ☐ error in judgement / incorrect decision
   ☐ poor communication
   ☐ failure to understand equipment
   ☐ failure to understand dive table
   ☐ poor maintenance of equipment
   ☐ malfunction or failure of equipment
   ☐ drug or alcohol intake
   ☐ lack of medical clearance to dive
   ☐ inadequate supervision
   ☐ lack of a buddy check

5. Did the incident occur whilst under training? Yes ☐ No ☐

6. What influence did the incident have on the dive plan?

   ☐ none ☐ delayed the dive
   ☐ aborted the dive ☐ changed the plan

7. Did the incident involve (you may need to tick more than 1):

   ☐ out of air situation
   ☐ rapid ascent
   ☐ omission of decompression stops
   ☐ misreading of decompression tables/computer
   ☐ multiple ascents/descents/bounce diving
   ☐ loss of buddy contact
   ☐ marine animal
   ☐ equalization problem on ascent
   ☐ equalization problem on descent
   ☐ giddiness/vertigo
   ☐ flying altitude after diving
   ☐ problem at safety stop.

8. Was the diver involved: a diving student ☐ untrained ☐ certified diver ☐

9. Diver certification level: basic instructor ☐ open water commercial ☐ advanced ☐ not known ☐ divemaster ☐

10. Sex: M ☐ F ☐ Diver's Age: ..... yrs. Not Known ☐

11. Which country did he/she train in: ☐ Not known ☐ AUS ☐ USA ☐ JAPAN ☐ NZ ☐ UK ☐ Other - specify

12. Please indicate geographical location of incident?

    ☐ Zone 1: Tropical Australia
    ☐ Zone 2: Lower W.A. & S.A.
    ☐ Zone 3: NSW, VIC, Tasmania
    ☐ Zone 4: Pacific
    ☐ Zone 5: New Zealand
    ☐ Zone 6: Other: specify .....

AIR SUPPLY

1. Air Consumption: ran low ☐ out of air ☐ not a problem ☐ buddy breathing ☐
   Octopus used ☐ not known ☐

2. If there had been an alternative air source would it have helped in the situation?

   Yes ☐ No ☐ Not known ☐

3. Regulator and air supply:

   ☐ didn't check contents gauge regularly
   ☐ air supply not turned on
   ☐ free flowing second stage
   ☐ contents gauge inaccurate/failed
   ☐ unable to read contents gauge at depth
   ☐ 1st stage problem
   ☐ problem with regulator despite frequent servicing
   ☐ hose rupture
   ☐ not involved

   City where normally serviced: .....
   Air consumption this dive greater than previous dives. Yes ☐ No ☐

BUOYANCY

1. Buoyancy problem: Yes ☐ No ☐

   ☐ overweighted
   ☐ underweighted
   ☐ air used frequently to maintain buoyancy.

2. Buoyancy Jacket:

   ☐ not worn
   ☐ inflation device failed
   ☐ inflation device not connected correctly
   ☐ unable to vent vest to slow down
   ☐ vest leaked
   ☐ vest provided inadequate buoyancy
   ☐ vest uncomfortable to wear
   ☐ unfamiliar with its use
   ☐ incorrect use
   Name of vest: .....
   Model of vest: .....
   ☐ Not involved.

DIVE TABLES/COMPUTER

1. Dive tables used:

   NONE ☐ USN ☐ RN ☐ BSAC/RNPL ☐ PADI ☐
   NAUI ☐ DCIEM ☐ BASSETT ☐ Other - specify: .....

2. Was a dive computer used? Yes ☐ No ☐

   If so: ☐ stopped working ☐ unable to read number
   ☐ inaccurate ☐ forgot to activate it

   Make: ..... Model: .....

GLEANINGS FROM MEDICAL JOURNALS

The following articles have come to the notice of the editorial staff and these notes are printed to bring them to the attention of members of SPUMS. They are listed, with their abstaracts, under various headings of interest to divers. Any reader who comes across an interesting article is requested to forward the reference to the Journal for inclusion in this column.

DIVING DROPOUTS

Diving dropouts: The Australian experience.
Wilks J. *Aust J Sci and Med in Sport* 1990; 23(1): 17-20

For a service-oriented industry like scuba diving the "dropout" problem represents considerable lost income. More importantly, it raises concerns about the adequacy of training and other factors which may contribute to people leaving the sport. In this study, 95 certified divers, who had not dived since completing their open water course, were questioned about their training, their reasons for dropping out, any frightening or unpleasant experiences, and their willingness to start diving again. Time constraints and economic factors were cited as the main barriers to participation. Training courses were rated favourably, and while there were some unpleasant experiences reported, 94 of the 95 respondents were interested to resume diving. Marketing strategies for encouraging greater participation in diving are discussed.

From

Key Centre in Strategic Management, Queensland University of Technology, Brisbane, Australia.

JELLYFISH STINGS

Granuloma annulare following bluebottle jellyfish (*Physalia utriculus*) sting.
Mandojana RM. *J Wilderness Med* 1990; 1: 220-224

This report describes the case of a teenager stung by a Pacific Portuguese Man-of-war, or bluebottle, jellyfish (*Physalia utriculus*) in Hawaii, who subsequently developed classical lesions of granuloma annulare. It is speculated that various toxic substances found in the complex venom of this particular coelenterate may be responsible for initiating the pathophysiologic mechanism.

Key words: Granuloma annulare, bluebottle, jellyfish sting.

From

Dermatology Associates of Knoxville, P.C, 930 Emerald Avneue, Suite 816, Knoxville, Tennessee 37917, U.S.A.

INTERATRIAL SHUNTS AND DECOMPRESSION SICKNESS IN DIVERS

Interatrial shunts and decompression sickness in divers
Smith DJ, Francis TJR, Hodgson M, Murrison AW and Sykes JJW. *Lancet*, 1990; 335: 1593
Letter

Stating that rapid onset spinal DCS is more likely to be due to authocthonous bubbles than to arterial gas emboli, but that in late onset spinal DCS and cerebral DCS arterial gas emboli are the more likely explanation.

From

Institute of Naval Medicine, Undersea Medicine Department, Alverstoke, Gosport, Hampshire PO12 2DL, UK.

Safety of contrast echocardiography in screening divers
James PB. *Lancet* 1990; 336:1389-90
Letter.

Suggests that the technique, which generates microbubbles, may not be valid or safe, because of false negatives and the risk of side effects.

From

Department of Community Medicine, Wolfson Institute of Occupational Health, University of Dundee, Medical School, Ninewells, Dundee, UK.

Safety of contrast echocardiography in screening divers
Cross SJ, Thomson LF, Lee HS, Shields TG and Jennings KP. *Lancet* 1990; 336: 1595-96
Letter (reply).

Risks of the procedure are low. Only using it on treated divers, (tissues are no longer nitrogen loaded) so embolised bubbles not expected to grow.

From

Department of Cardiology, Aberdeen Royal Infirmary, Fosterhill, Aberdeen AB9 2ZD, UK, and Hyperbaric Medicine Unit, Aberdeen Royal Infirmary.

SELECTED ABSTRACTS

Reprinted from the Program and Abstracts of the UNDERSEA AND HYPERBARIC MEDICAL SOCIETY ANNUAL SCIENTIFIC MEETING 6-11 June 1989

The address of the Undersea and Hyperbaric Medical Society, Inc. is 9650 Rockville Pike, Bethesda, Maryland 20814, U.S.A.

DIVING DEATHS

A review of 25 diving fatalities in British Columbia 1983 - 1988.

Buckingham I. *Undersea Biomed Res* 1989; 16 (Supp): 33

As a local coroner, the author has investigated 25 diving fatalities over six years, 1983-1988 inclusive. Twenty one cases were autopsied and death was 57% due to asphyxial drowning. Forty-three per cent were due to arterial gas embolism following pulmonary inflation. The activities pursued while diving revealed 56% due to recreation, 36% commercial and 8% military. A review of the deaths points out that many mistakes were made by the divers who ignored the most basic diving rules. Thirty-six per cent of the victims were diving alone so that no immediate assistance was rendered. Eighty per cent of the victims died using scuba equipment, 12% hookah and 8% closed circuit devices. Those using hookah were principally on their first dives with the equipment, untutored and alone in their death. The closed circuit deaths involved dilutional hypoxia and oxygen toxicity. Several divers were overweighted which led to rapid fatigue and further difficulty. Examination of the buoyancy compensators showed a surprising number of problems. Several compensators had their inflation cartridges removed and those who had cartridges frequently had them incorrectly fitted. Findings from these accidents have been contributory to changing the laws of the Workers Compensation Board in the Province. Sports trained divers are no longer allowed to directly enter the commercial diving field without some prior training.

2580 Lansdowne Road, Victoria, B.C. V8R 3P3, Canada.

REPETITIVE DIVING

Doppler ultrasound monitoring of gas phase formation and resolution in repetitive diving.

Powell MR and Rogers RE. *Undersea Biomed Res* 1989; 16 (Supp): 69

In an experiemental series of multi-day (6/day), repetitive dives (4/day), it was found that multi-level dives produced the smallest Doppler-detectable gas phases as compared with square-wave dives. This is hypothesized to be related to minimal gas phase formation during the decompression phases. There was an indication that the interval between dives was important (aside from obvious gas elimination), and this is postulated to be related to the time necessary to resolve the formed tissue gas phase.[1] When the surface interval becomes short, total gas elimination in micro regions can be less than expected from the basic Haldane algorithm. This has been treated in decompression tables by the addition of long half-time compartments to handle either a real or a virtual condition.

1 Griffiths, H.B. et al. *Proc Roy Soc B* 1971; 178: 389

Institute of Applied Physiology and Medicine, Seattle, WA 98122, U.S.A. and Pensacola, FL, U.S.A.

Comparison of proposed new sport diving tables with Navy standard air decompression tables using tissue M values.

Bookspan J and Bove AA. *Undersea Biomed Res* 1989; 16 (Supp): 66

Recently published diving tables (PADI) for sport divers allow longer bottom times in repetitive and multilevel diving. Validation studies of these tables did not compare them to established tables, making results difficult to interpret. New tables shorten surface interval (SI), lengthen repetitive dive (RD) time and shorten first dives. Based on 60 minute rather than 120 minute controlling tissue. The increased bottom time and decreased surface interval differ from accepted US Navy tables. To evaluate the affect of these changes, we compared tissue nitrogen pressure (calculated courtesy Inst. for Env. Med., University of PA) in 12 halftime tissues (as % of M value) after four comparable RD profiles: two two-dive USN and PADI profiles, and two four-dive USN and PADI profiles. All profiles were to prescribed limits. Results indicate:

1. Lower nitrogen uptake following the first PADI dive was due to the shortened bottom time (dive 1 %M of 40 min tissue - PADI: 89±0.6, USN: 96±0.8, $P<0.2$).
2. For dives 2-4, the USN RD profiles resulted in lower nitrogen tensions than PADI RD profiles in tissues with halftimes greater than ten minutes (dives 2-4 %M of 40 min tissue PADI: 96±0.8, USN: 90±2.0, $P<0.001$).
3. Highest tissue loading occurred in the 40 minute and not the 60 minute tissue. Loading of the 40 minute tissue may indicate that longer bottom times and shorter SI are not recommended in RD. If relative safety of diving tables is predicated on lower tissue nitrogen pressure, USN tables are likely to be a safer choice in repetitive profiles.

Temple University Hospital, Philadelphia, PA 19140, U.S.A.

DECOMPRESSION SICKNESS

Somotosensory evoked potentials measured in divers with a history of spinal cord decompression sickness.
Overlock R, Dutka A, Farm FJnr, Okamoto G and Suzuki D. *Undersea Biomed Res* 1989; 16 (Supp): 89

For several years the Naval Medical Research Institute (NMRI) has conducted animal studies of decompression sickness using animal models and measuring somatosensory evoked potentials (SEP). This study was undertaken to compare the sensitivity of SEP measurements with that of a careful neurological evaluation in human subjects with varying degrees of residual deficit from decompression sickness of the spinal cord. Twenty-three divers were grouped by degree of disability. Four were wheelchair bound since their accident and had Kurtzke's Disability Status Scale (DSS) scores of 7; nine had obvious neurological deficits with DSS scores of 2 to 5; six had only subtle or no neurological findings with DSS scores of 0 to 1; four normals were included for comparison of SEP results. Results of neurological examination were very consistent with definite spinal levels as expected with spinal injury. SEP latency measurements were not consistently recorded at spinal levels L5, T10, and C6, but were repeatable at cortical levels. Tibial to Cortical latencies were considered the most reliable SEP measures and showed significant difference in only the wheelchair (DSS=7) group where they were absent. All other groups had mean Tibial-Cortical latencies between 37.3 and 42.4 milliseconds. We conclude that SEP studies may be helpful only in very severe residual decompression sickness and that careful neurological examination is a more sensitive measure of residuals.

Hyperbaric Treatment Centre, Honolulu, Hawaii, U.S.A.; Naval Medical Research Institute, Bethesda, Maryland 20814, U.S.A.; Rehabilitation Hospital of the Pacific, Honolulu, Hawaii, U.S.A.

GAS PHYSIOLOGY

Dyspnea and endtidal PCO_2 as criterial of acceptable breathing resistance in diving gear.
Warkander DE, Nagasawa GK, Norfleet WT and Lundgren CEG. *Undersea Biomed Res* 1989; 16 (Supp): 95

This project is seeking physiological design criteria for divers' breathing gear. Six prone subjects were, in a wet pot, exposed to standardized leg exercise for 25 minutes at 60% of their maximum O_2 uptake at 1.45 ATA (15 fsw) and 6.8 ATA (190 fsw). The subjects wore a full face mask which was connected to a bag-in-box system. The breathing gas was air. Annular resistance discs were inserted in the breathing circuit inducing breathing resistance at three levels (high, moderate, low) causing peak inspiratory to peak expiratory pressure excursions of 40-45, 30-35, 15-20 cm H_2O, respectively. Control experiments were at 5-10 cm H_2O. The subjects reported dyspnea according to a three tiered scale (non-moderate, severe). Endtidal gas composition and mask pressure excursions were recorded. Dyspnea scores tended to increase with respiratory pressure excursions and no dyspnea was reported at excursions less than 15 cm H_2O at 1.45 ATA and 10 cm H_2O at 6.8 ATA. Entidal CO_2 concentrations showed small increases with increased resistance but were considerably more influenced by depth, being higher at 6.8 ATA. Importantly, in some subjects hypercapnia was combined with lack of dyspnoeic sensations despite high breathing resistance. It follows that neither dyspnea nor endtidal CO_2 levels can be used as sole indicators of acceptable breathing resistance in diving gear.

Center for Research in Special Environments, Department of Physiology, School of Medicine, SUNY, Buffalo, NY 14214, U.S.A.

Gas density limitations to inspiratory flow.
Miller JN. *Undersea Biomed Res* 1989; 16 (Supp): 94

Inspiratory flow, unlike expiratory flow, is ordinarily limited by inspiratory effort. An imposed external resistance may limit inspiratory flow, be it a poorly designed breathing apparatus or laryngospasm caused by the inadvertent inhalation of foreign material. Persistent efforts to inhale against a significant external resistance can result in acute pulmonary edema. Inspiratory and expiratory isovolume pressure curves were constructed from inspired volume, transpulmonary pressure and inspiratory flow data recorded from multiple inspiratory manoeuvres in two subjects at a variety of different gas densities (1.29, 2.30, 5.13, 7.70 and 10.01 $G.L^{-1}$, equivalent to one, two, four, six and 7.8 ATA compressed air) in a high pressure chamber. Maximal ranges remained effort-dependent at all gas densities, while inspiratory flow transitioned from effort-dependent to effort-independent at low lung volumes at gas densities above 7.70 $G.L^{-1}$ (6 ATA breathing air). The lung volume at which this transition occurred increased at higher gas densities forcing effort-dependent inspiratory flow into the upper lung volume range, requiring further effort to overcome the reduction in lung and chest wall compliance found in the upper 50% of vital capacity. Sustained inspiratory effort appeared to peak at respiratory transpulmonary pressures in the range of -20 cmH_20, effectively limiting inspiratory work of breathing. This combination of breathing at high lung volumes against decreased pulmonary and chest wall compliance and physiological limitations to inspiratory work, can be expected to be further limited by the imposition of breathing equipment of high inspiratory resistance.

Department of Anesthesiology, University of South Alabama, 2451 Fillingim Street, Mobile, AL 36617, U.S.A.

HYPERBARIC OXYGEN

Mechanism of decreased coronary and systemic blood flow during hyperbaric oxygenation.
Savitt MJ, Elbeery JR, Owen CH, Rankin JS and Camporesi EM. *Undersea Biomed Res* 1989; 16 (Supp): 50

It is well known that hyperbaric oxygenation (HBO) decreases coronary blood flow (CBF) and cardiac output (CO), but the mechanism has not been defined. In order to determine whether this is a primary myocardial phenomenon, ten chronically instrumented conscious dogs were studied during pharmacologic autonomic blockade. Data were recorded during control conditions and during multiple partial vena caval occlusions, at 1 ATA breathing air, and at 3 ATA on 100% O_2. Oxygen was administered at pressure with a head-tent assembly. At 3 ATA PaO_2 increased from 86±5 to 1374±201 mmHG (means±SD), while $PaCO_2$ and pH values were not statistically different. At 3 ATA, arterial O_2 content increased from 13±4 vol% to 17±1 vol%, CS O_2 content increased from 4±0 vol% to 10±1 vol%, CO decreased by 21±14%, CBF fell by 17±10%, and myocardial O_2 consumption (MVO_2) decreased by 11±6% (all p,0.05); [A-CS]DO_2 was unchanged (p=0.10). In these autonomically blocked animals, HR did not decrease significantly at 3 ATA (from 112±19 to 106±22 bpm). Intrinsic myocardial function, as measured by the stroke work-end diastolic volume relationship, and myocardial energetics, as determined by the linear relationship between total myocardial oxygen consumption and total mechanical energy expenditure (LV stroke work + LV pressure - volume product) were both unchanged from 1 ATA to 3 ATA O_2 (p >0.20). Thus, the diminished CBF and CO with HBO do not appear to be associated with primary alterations in myocardial energetic function.

Depts of Surgery, Anesthesiology, Cell Biology and the Hyperbaric Center, Duke University, Box 3823, Durham, N.C. 27710, U.S.A.

Hydrogen sulphide poisoning and the use of hyperbaric oxygen; a report on six cases.
Sheeran S, Pearson K and Kerr D. *Undersea Biomed Res* 1989; 16 (Supp): 21

Hydrogen sulphide poisoning and cyanide poisoning may have similar mechanisms of toxicity. Research has indicated that there is a role for the use of hyperbaric oxygen (HBO) in the treatment of cyanide poisoning. It is believed that there is an indication for the use of HBO in the management of hydrogen sulphide poisoning victims. This report describes the aetiology of exposure, physiological effects, initial management at the site of the accident, and the resulting management in the multiplace hyperbaric chamber at the Prince Henry Hospital. It should be emphasized that the time elapsed between the toxic exposure and treatment with HBO is critical. Four of the six cases were transported from Darwin in the Northern Territory which is approximately 3200 kilometres (1987 miles) from Sydney, with a time delay of 36 hours. Two of these victims died as a result of injuries sustained during the accident. The other two cases were retrieved from a local tannery and treated within 45 minutes. Their recovery was relatively uneventful. The clinical course of these six cases provides good evidence that HBO is useful in the management of hydrogen sulphide poisoning.

Department of Diving and Hyperbaric Medicine, The Prince Henry Hospital, Anzac Parade, Sydney NSW 2036, Australia.

Hyperbaric treatment of iatrogenic air embolism.
Massey EW, Shelton DL, Moon RE and Camporesi EM. *Undersea Biomed Res* 1989; 16 (Supp): 25

Air embolism (AE) may occur as a rare complication of surgical procedures. We report 14 patients (7 female) with iatrogenic AE. Age ranged from 5 to 84 years (mean 46.9 years). Seven patients had onset of deficit in the immediate perioperative period: 5 had cardiac surgery, 2 had neurosurgical procedures; 4 had invasive radiologic procedures; 1 was on dialysis and 2 cases were associated with jugular vein catheterization. All iatrogenic air embolism patients receiving hyperbaric oxygen therapy (HBO) at Duke University during the last 9 years were entered into this retrospective study. Clinical presentation included coma, diplopia, hemiparesis, seizures: most commonly a sudden focal neurologic deficit indistinguishable from other cerebral stroke syndromes. Distribution involved the middle cerebral artery in 8 patients and multiple vessels in 6 patients. In one case neurological exam had returned to normal by the time of presentation at the hyperbaric chamber. Most patients were treated with Table 6 or 6A, with extensions as required. Neurological examination was done pre- and post-therapy. Delay between AE and treatment ranged from 1.2 to 42 hours (mean 17.5 hours). Outcome included 3 deaths; 2 unchanged (= total of **5** patients not improved; mean treatment delay 19.9 ± 14 hour [SD]); 6 had major improvement by neurologic examination and in symptoms; 2 had minor improvement and 1 had complete resolution (= total of **9** patients improved: mean treatment delay 16.1 ± 13.3 hr). The difference in delay times between the two groups was not statistically significant. Delay in treatment of 5 patients was >24 hours. Two of the patients who had major improvement with HBO were treated at 31 and 42 hours after AE. We recommend that delay in referral should not discourage hyperbaric treatment.

Departments of Medicine, Anesthesiology and Cell Biology, and Hyperbaric Center, Box 3823, Duke University, Durham, N.C. 27710, U.S.A.

Hyperbaric oxygen therapy reduces mortality and debridements for necrotizing fasciitis.
Riseman JA, Zamboni WA, Curtis A, Konrad HR and Ross DS. *Undersea Biomed Res* 1989; 16 (Supp): 81

Twenty six patients with necrotizing fasciitis were treated from 1983 to 1988. Two groups of patients were established in this review: Group 1 (n=9) received surgical debridement and antibiotics only; Group 2 (n=17) received HBO (90 minutes at 2.5 atmospheres, average 7.4 treatments) in addition to surgery and antiobiotics. Both groups were similar in age, race, sex, wound bacteriology and antiobiotic regimen. Body surface areas affected was similar; however, perineal involvement was more common in Group 2 (53%) vs. Group 1 (12%). Although Group 2 patients receiving HBO were more critical on admission, mortality was significantly lower (22%) compared to Group 1 (66%) ($p<0.05$). In addition, only 1.2 debridements per Group 2 patient were required to achieve wound control versus 4.0 debridements per Group 1 patient. The addition of GBO therapy to the surgical and antiobiotic treatment of necrotizing fasciitis significantly reduced mortality and wound morbidity (number of debridements) in this study. These results strongly support the routine use of HBO in treating this condition.

Southern Illinois Uni. School of Medicine, 800 North Rutledge, Springfield, IL 62781, U.S.A.

Gas gangrene: a ten year experience at Toronto General Hospital.
Ellis BJ and Koch GH. *Undersea Biomed Res* 1989; 16 (Supp): 79

The Hyperbaric Department at Toronto General Hospital is a hospital-based facility with a referral population of about seven million people. During the ten year period January 1, 1979 to December 31, 1988, fifty cases of suspected gas gangrene were treated. Forty-eight of these cases were reviewed. All (34 males and 14 females) were treated with a combination of antibiotics, surgery and hyperbaric oxygen, which remains the treatment of choice. The order of surgery first versus HBO first has not yet been established, though anecdotes suggest HBO first leads to less tissue loss (i.e. amputation). A prospective study needs to be done to clarify this. The overall mortality was 22.9% with a 33.3% amputation rate. Nineteen of the cases (39.5%) were secondary to trauma while 17 (35.4%) were post-operative and 14 (29.1%) fit neither category. The mortality rate in the groups varied widely. The trauma group had only one death (5.3%), the post-operative group three deaths (17.6%), while the mortality rate for the group of other cases was 50%. Factors contributing to increased mortality included pre-existing diabetes, advanced age and the location of the infection. At the same facility between 1960 and 1970 there were also fifty cases of suspected gas gangrene of which 18 (36%) were secondary to trauma, indicating no change in statistics over a twenty year period.

Hyperbaric Department, Toronto General Hospital, 200 Elizabeth Street, Toronto, Ontario, Canada.

Prognostic value of brain CT after HBO in severe CO poisoning.
Fife CE, Sallee DS, Gray L and Piantadosi CA. *Undersea Biomed Res* 1989; 16 (Supp): 19

Hyperbaric oxygen is the treatment of choice in severe carbon monoxide (CO) poisoning. Characteristic brain lesions after CO poisoning particularly involving the basal ganglia, have been described by CT but the prognostic value of such studies has not been determined in patients treated with hyperbaric oxygen (HBO). Fifteen patients with serious carbon monoxide poisoning, presenting with coma, semi-coma or severe mental status changes who received HBO therapy were evaluated with CT brain scans. Clinical outcome was analyzed retrospectively and correlated with CT findings described by a neuroradiologist blinded as to outcome. Patients ranged in age between 19 and 73 years. Sources of CO included: motor vehicle exhaust (9) including four suicide attempts, house fires (3), and home heaters (2). CO levels ranged from 0.4% to 58% at presentation one to 24 hours post exposure; all treated with HBO at pressures of 46.2 fsw for 90 minutes or in one case 68 fsw for 2 hours. If necessary, subsequent treatments at 33 fsw for 2 hours were administered until patients recovered or until no further neurological improvements were observed. Total number of treatments ranged from one to ten with a mean of three treatments per patient. Of the 15 patients, six had abnormal CT scans (40%) and one scan was equivocal. All positive scans were in patients with a history of coma. CT abnormalities consisted of either basal ganglia infarcts (5) or diffuse cerebral edema (1). All patients with positive CT scans had poor outcomes including: death (2), Parkinsonism and dementia (1), impaired language function and hearing loss (1), severe personality changes (1) and partial blindness (1). Of the 9 patients with normal or equivocal CT scans, 7 of whom had a history of coma, none had permanent neurological sequelae after treatment with HBO. Thus, patients with CO poisoning who had positive CT scans after HBO had uniformly poor outcomes despite aggressive HBO. However, severely poisoned CO patients with negative CT scans had uniformly good outcomes when treated with HBO despite a history of coma.

F.G. Hall Hypo-Hyperbaric Center, Duke University Medical Centre, Durham, N.C. 27710, U.S.A.

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SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY (SPUMS)

DIVING EMERGENCY SERVICE (D.E.S.) RAFFLE

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1. 7 day/6 night dive expedition from Townsville aboard "Spoilsport" (donated by Mike Ball Dive Expeditions)* (*Prize is for one person, is not transferable and does not include airfare, bar bill or equipment hire. Must be taken Jan-June 1992)	**Value = $1866**
2. Scubapro Mk10 G250 regulator (donated by Scubapro Australia)	Value = $599
3. U.S. Divers Conshelf SE2 regulator (donated by Aqualung Australia)	Value = $460
4. U.S. Divers Conshelf SE2 regulator (donated by Aqualung Australia)	Value = $460
5.. Sea Hornet Command Air regulator (donated by T.D. Preece &Co)	Value = $450
6. Seiko 200m diver's watch (donated by Seiko Australia)	Value = $450
7. "Chatty Crab" underwater communication device (donated by Cape Byron Imports)	Value = $428
8. Digital Combo 2 Deluxe gauges (donated by Sterling Leisure Industries)	Value = $416
9. Sonar tailor made wetsuit - 5mm or 7 mm, as requested by winner (donated by Sonar Wetsuits)	Value = $400
10. 85 cu. ft cylinder (donated by CIG)	Value = $360
11. Oceanic Omega octopus regulator (donated by Oceanic Diving Australia)	Value = $275
12. Delta Octo Plus (donated by Divers Supplies Australia)	Value = $195
13. 1 pair of Diving Star DF7 fins (donated by Scuba Industries of Australia)	Value = $135
14. 1 pair of Diving Star DF7 fins (donated by Scuba Industries of Australia)	Value = $135
15. 1 pair of Diving Star DF7 fins (donated by Scuba Industries of Australia)	Value = $135
16. Apollo mask and snorkel (donated by Apollo Australia)	Value = $100

A raffle, under the auspices of SPUMS, has been organised by John Lippmann to raise money for the Diver Emergency Service. Above is a copy of a ticket showing the prizes, which in total are worth over $ 6800. The diving industry has made a great contribution and it is up to divers to show their support. Tickets (only 3,000 available) at $ 5 each can be purchased at SCUBA Expo and at certain dive shops. Tickets can also be purchased by sending a cheque or money order for $ 5, with a stamped self-addressed envelope to: DES Raffle, PO Box 381, Carnegie, Victoria 3163. The draw is on September 19th 1991. SEND FOR YOUR TICKET AND SUPPORT THE DIVER EMERGENCY SERVICE.