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South Pacific Underwater Medicine Society Incorporated

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GLEANINGS FROM MEDICAL JOURNALS, COURSES AND MEETINGS ARE DETAILED ON THE BACK COVER

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OBJECTS OF THE SOCIETY

To promote and facilitate the study of all aspects of underwater and hyperbaric medicine.
To provide information on underwater and hyperbaric medicine.
To publish a journal.
To convene members of the Society annually at a scientific conference.

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

The 1998 subscription for Full Members is $A100.00 and for Associate Members is $A 50.00.

The Society's financial year is January to December, the same as the Journal year.

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The printed copies should be double-spaced, using both upper and lower case, on one side of the paper only, on A4 paper. Headings should conform to the format in the Journal. 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 double spaced on separate sheets of paper. **No vertical or horizontal rules are to be used.**

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.

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 illness (DCI) can thereafter be referred to as DCI.

The preferred length for 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. Accuracy of the references is the responsibility of authors. Acknowledgments 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).

References

The Journal reference style is the "Vancouver" style, printed in the Medical Journal of Australia, February 15, 1988; 148: 189-194. In this system 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

There should be no full stops after the reference numbers. There should be a space after the semi-colon following the year and another after the colon before the page number and no full stop after the page numbers. The Journal uses two spaces after a full stop and before and after the journal name in the reference. The titles of books and of quoted journals should be in italics.

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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.

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GLEANINGS FROM MEDICAL JOURNALS

A superior heart de-airing technique reduces cerebral arterial gas embolism following left heart valve surgery — Milsom P and Mitchell SJ — 234

MEETINGS AND COURSES

204, 234 and 239

DIVER EMERGENCY SERVICE
PHONE NUMBERS
1-800-088-200 (Australia)

+61-8-8373-5312 (International)

The DES number 1-800-088-200 can only be used in Australia.
For access to the same service from outside Australia ring ISD 61-8-8373-5312.

PROJECT STICKYBEAK

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

Information may be sent (in confidence) to:

Dr D. Walker, P.O. Box 120, Narrabeen, N.S.W. 2101.

DIVING INCIDENT MONITORING STUDY (DIMS)

DIMS is an ongoing study of diving incidents. An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report any incident occurring in your dive party, but do not identify anyone. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver damage. Using this information to alter diver behaviour will make diving safer.

To obtain Diving Incident Report forms write to

DIMS,
GPO Box 400, Adelaide, South Australia 5000.

PROJECT PROTEUS

The aim of this investigation is to establish a data base of divers who dive or have dived with any medical contraindications to diving. At present it is known that some asthmatics dive and that some insulin dependant diabetics dive. What is not known is how many. How many with these conditions die is known. But how many dive safely with these conditions is not. Nor is incidence of diving accidents in these groups known.

If you are in such a group please make contact. All information will be treated as CONFIDENTIAL. No identifying details will appear in any report derived from the data base.

Write to

Project Proteus
PO Box 120, Narrabeen, New South Wales 2101, Australia.

The Editor's Offering

A Happy Christmas and a Prosperous New Year to all Members, Associates and readers. The Editor's thanks go to Drs John Couper-Smartt and David Davies who had once again provided the proof reading needed to reduce the typographical errors and convoluted syntax which get published, in error, in the Journal. Thanks are also due to those who provide peer review for papers submitted to the Journal. The Editor feels that, as the reviews are only disclosed to authors without naming the referee, referees' names should be a secret.

This year the Society had its Annual General Meeting in New Zealand for the first time. Dr Mike Davis was the convener and he produced a magnificent meeting. The workshop theme was The initial management of diving accidents. The workshop showed that many people were not aware that decompression illness is not the only cause of injury to divers. Nearly drowning, near drowning and drowning affect a much larger group of divers. Drowning kills 56% of scuba diving deaths while decompression illness (cerebral arterial gas embolism or CAGE) kills 14%. Decompression sickness does not figure as a cause of death in the 177 Australian scuba divers who died between 1972 and 1993. Forty nine percent of these divers had low or no air remaining. Any protocol for early management of diving accidents must take into account that many rescued divers will have inhaled water, which is associated in most cases with hypoxia and, in many, unconsciousness.

The Society's policy on *The initial management of diving injuries and illnesses* is printed on page 193. SPUMS flow charts for *Missed Decompression Procedures* and *Diving Accident Procedures* appear on pages 197 and 200 respectively. Between them are reprints of three flow charts for Diving Accident First Aid published in the SPUMS Journal in the early 1980s. All readers should study the policy and the flow charts. Do you dive with the precautions SPUMS recommends?

This issue deals with the lung and diving, its physiology and the changes and dangers of immersion and ascent. Besides an excellent review of pulmonary barotrauma (PBT) by James Francis there are papers by Richard Moon (physiology), Chris Lawrence (gas at autopsy in divers) and Simon Mitchell (counting arterial gas emboli during open heart surgery and studying mental changes after such surgery). Dr Francis draws attention to the frequency of CAGE in divers while it is rare in all other cases of PBT. Overenthusiastic positive pressure ventilation causes pneumothorax or, very often, pneumothoraces but not CAGE.

In early 1955 the Editor, as a newly joined Surgeon Lieutenant RN, attended the 100 ft tower (submarine escape training tank) at HMS DOLPHIN to see how it was done. The facility had been open about a year and a number of cases of CAGE had been diagnosed and treated, mostly successfully once the recompression chamber had been resited to the top of the tower. Diagnosis was simple. Every trainee who arrived at the surface conscious climbed out and stood against the wall for five minutes. Anyone who collapsed was seized and inserted into the chamber, rather like a shell into the breech of a gun. An attendant was already waiting in the chamber, all through the session, and the young doctor followed at the heels of the casualty. This day the trainee regained consciousness as the chamber pressurised towards 165 ft (50 m), as did the vast majority of the casualties. However he became acutely short of breath on the way up. The doctor inside the chamber confirmed the diagnosis of a tension pneumothorax by percussion and the Editor and another new doctor were sent across the road to the Royal Naval Hospital, Haslar (about 400 m) to collect a chest drainage set. This was the first pneumothorax which had occurred in the tower. Some twenty minutes after we got back with the chest drain etc. we were on our way back to Haslar for another for the diver had two tension pneumothoraces, each becoming evident at a different depth. The medical staff at the tower and at HMS VERNON (the diving school) could not recall ever having seen a pneumothorax in a diver. Readers are referred to page 214 for the reasons.

In The World As It Is we report the first known Australian attempt at line dancing underwater while wearing the old standard dress. By chance two articles about early diving equipment have recently been published in the British magazine *Diver*. On page 253 is the story of a self contained underwater breathing apparatus (scuba) made and used in France in 1828. That is not a typo, we mean the early 19th century. The other on page 237 is about the sponge divers of the Greek island of Symi, whose diving habits were similar to those of the pearl divers of Broome, described in the Pearl Diving Supplement (*SPUMS J* 1996; 26 (Suppl):), and at least as lethal.

The Editor cannot send his readers each a Christmas card but can reproduce the cartoon from the cover of the October to December 1979 Journal.

ORIGINAL PAPERS

DROWNING SYNDROMES WITH SCUBA

Carl Edmonds, Douglas Walker and Briony Scott

Abstract

Because of the lack of detail available on the aspiration syndromes associated with scuba diving, two surveys were conducted and compared.

The first survey involved 100 scuba deaths attributable to drowning in the Australian dive fatality series (Project Stickybeak). The second survey involved a questionnaire sent out to divers on the internet who had aspirated and "nearly drowned". There were 48 questionnaires suitable for coding.

The conclusions from the two surveys, and comparisons between them, reveal; the importance of personal diving practice including both medical and physical fitness, the value of experience in undertaking diving operations, faulty equipment and misuse of equipment, the effects of hazardous environments, the value of neutral buoyancy being maintained during the dive and not being dependent upon the buoyancy compensator, as far as possible.

Other factors which increased the likelihood of problems developing and inducing an unsuccessful outcome included; an inadequate air supply, the failure to employ correct buddy diving practices, the failure to become positively buoyant after the start of a diving incident, possible inadequate buddy communication and inappropriate or delayed rescue and resuscitation.

Key Words

Accidents, deaths, drowning, incidents, near drowning, salt water aspiration

Background

A normally functioning human, with adequate equipment in a congenial ocean environment, is protected from drowning as he carries his own personal life support with him, his own air supply. Drowning would only happen when there is; diver error, failure of the equipment to supply air or hazardous environmental influences.

Nevertheless, the commonest ultimate cause of death in recreational scuba divers is drowning. Factual information that clarifies the causes and management of drowning, could be of value in preventing further fatalities.

Previous surveys illustrated the importance of drowning as the ultimate cause in 74-82% of recreational scuba diving fatalities.[1-5] It contrasted with the rarity of the more conventionally accepted diving diseases, decompression sickness and gas contamination, which each accounted for less than 1% of the deaths.

Those surveys also demonstrated the demographic similarity of the USA and Australia recreational diving deaths. This observation is understandable as they have a similar socio-economic standing, are controlled by the same diver instructor organisations and use the same diving equipment.

Of note, in the more detailed surveys, was the high frequency of multiple contributing factors to each death.[3-5] The diagnosis of drowning tended to obscure those preceding factors. The drowning sequelae and drowning pathology were due to the environment in which the accident occurred, not the initiating or primary cause of death.

For example, loss of consciousness when engaging in terrestrial activities is unlikely to cause death. Under water death often follows unconsciousness.

The three major manifestations of inhalation of water in the scuba diving are:

1 Aspiration syndromes (causing symptoms and signs)
2 Near drowning (producing unconsciousness)
3 Drowning (causing death).

The aspiration of sea water causing clinical features in scuba divers, who retain consciousness, has been dealt with in another paper.[6] Sometimes aspiration progresses to the other manifestations of near drowning and drowning.[3,6]

Specific attention to both the scuba drowning deaths and the "nearly drowned" have been poorly documented in the literature. Some texts on diving medicine hardly mention it. For this reason the following retrospective analyses were undertaken and compared.

Surveys

DROWNING SURVEY (DEATHS)

The last 100 scuba deaths attributable to drowning in the Australian Dive Fatality Series (Project Stickybeak) were reviewed. The following criteria were required for admission:

Compressed air diving equipment had to be worn by the victim in the water, with the intent of diving;

Exclusion of all military, large commercial or helium diving activities;

At least 3 of the 4 following sources of detailed information;

A Coroner's Inquest of Inquiry (full transcript including witness declarations and cross-examination);

Autopsy findings (anatomy, histology and toxicology in detail);

Official Government (Navy, Water Police etc.) assessment of equipment functioning and in-water trials. This included gas analysis of scuba tank compressed air;

Detailed written accounts of witnesses (buddies, other divers, boatmen or bystanders, rescuers).

All cases with the above criteria and designated as drowning in Project Stickybeak were included. These mainly covered a period over the last decade, although it had to be extended to obtain some data from the preceding decade, to as far back as the early 1980s.

NEAR-DROWNING AND ASPIRATION SURVEY (SURVIVORS)

Diving organisation, both national and international, which had previously promoted themselves as collecting diving accident questionnaires, were approached for assistance.

With one exception, they could give no information or supplied data with insufficient detail to be of any value. PADI (Australia) did supply valuable data on 12 divers, but too late to include in this paper. It will be coded as part of an ongoing study of this condition.

So a new survey was carried out, using various message boards and web sites and organisations on the Internet, during the first three months of 1997. We guaranteed anonymity to the respondents, although the names of the respondents were given in all but one case.

A questionnaire entitled "Have you nearly drowned?" was submitted to Web sites that were designed for divers. To increase compliance the questionnaire was kept to 2 pages, but it was designed to supply most of the information we required. If the diver wished, there was the option of supplying more information.

Forty-eight replies were able to be coded, some others were either not comprehensive enough to include or not pertinent. Only completed questionnaires were used.

Demographics

The average age of the survivor group was slightly older than the fatalities, probably reflecting the fact that survivors, by definition, have added some time to their post-incident life span.

Of the 100 fatalities, 89% were male, 11% female. During this period the female frequency amongst divers varied from 25- 35% depending on the years involved and authority sought. Of the 48 survivors , 52% were male and 48% female.

Compared with the diving population at the time, it appears that males are over represented in the scuba drowning cases, as they are in almost all other forms of drowning.[6] The surprise was that females appeared to be over represented in the "survivor" series. Whether females had more accidents, or whether they only reported them more frequently, could not be deduced. However, it does appear as if the female accidents result in fewer deaths. The gender response on the Internet (survivor) survey also was a surprise. We had presumed, in common with most other marketing groups, that the Internet would be dominated by males.

Personal factors

TRAINING

In the fatalities, 54% had completed scuba training but a high 38% had no known formal qualification. This group were approximately equally divided between:

a those in whom the documentation was inadequate;

b those without training, but who were experimenting with scuba on their own or with their friends;

c those who were engaged in introductory dives, brief resort courses or "dive experiences" with a recognised commercial organisation.

Of the survivors 81% had completed training and only 4% had no formal qualification. A surprising number in both groups were under formal training at the time, 8% of the fatalities and 15% of the survivors.

EXPERIENCE

This did not entirely correlate with training. In both the fatality and survivor series, the divers were equally represented amongst inexperienced (< 5 dives), novice divers (usually 5-20 dives) and experienced divers, one third each.

Of the fatalities, over one half were experiencing diving situations to which they had not been previously exposed, and only one third had previous experience of the conditions in which they died. The others were unable to be assessed.

The buddy or dive leader appeared to be considerably more experienced than the diver in most cases,

possibly explaining why the diver died and the buddy survived.

The survivor questionnaires were not appropriate for us to compare the relative experience of the divers to undertake the eventful dive.

TABLE 1

Experience	**Fatalities**	**Survivors**
Nil or slight	37%	31%
Novice	30%	35%
Experienced	27%	29%
Very experienced	6%	4%
Experienced enough to under take the dive		
No	56%	
Yes	32%	

VICTIM'S BEHAVIOUR

In those fatalities that were observed (to achieve 100 cases we had to extend the survey beyond the last 100, because of the number of "solo" divers) over a third were noted to have either a panic response or rapid/abnormal movements.

Over half the survivors reported these sensations. The increased incidence of panic and rapid or abnormal movements in the surviving group could well be attributed to the fact that this is a reported sensation, whereas the fatality figure represented the observed behaviour. Panic could well have been experienced, but not necessarily observed in the latter group. Over half the fatalities showed no change in their behaviour, with loss of consciousness being the first objective warning in a third. Loss of consciousness was the first manifestation noted in a quarter of the survivors.

Of interest was the absence of panic in many of the cases, during the development of the incident, even though it is a frequent initiator of other diving deaths.[1-3,6] Drowning scuba divers frequently drown quietly, possibly because of the effects of previous aspiration (hypoxia), depth (narcosis) or training ("don't panic", like the "drown proofing" of babies).

Twenty one percent of the fatalities requested assistance, in the form of an air supply. It was difficult to judge the success of this in many instances, as although they were sometimes reported as successful, the subsequent events would indicate otherwise. A request for a supplementary air supply was made by twice as many fatalities as survivors. This may bring into question the value of relying on a buddy responding to such a request. Alternatively, as we will see later, it is more frequent for the buddy to offer an emergency air supply, and this may be appreciated.

Occasionally there was the apocryphal underwater tussle for a single regulator. When the "low on air" (LOA) diver went for an air supply, he more frequently sought the companion's primary regulator than the octopus.

TABLE 2

Victim's behaviour	**Fatalities** (Observed)	**Survivors** (Reported)
Panic	21%	27%
Rapid/abnormal movement	16%	31%
Nothing unusual	63%	42%
Loss of consciousness	33%	25%
Air requested	21%	10%

MEDICAL DISORDERS

This is a contentious area, not only regarding the incidence of medical disorders but also their significance. Authors differ in their assessments of this[3,6,7] and none are free of prejudice.

Project Stickybeak is the most comprehensive data base of diving fatalities available. It is compiled by Douglas Walker in Australia and has been running for 25 years. Unfortunately, any medical history obtained from these records is inevitably an underestimate. In one analysis originally based on such data,[3] when an attempt was made to search for and complete the medical history, in less than half of the cases could this be achieved. Even then, it was often not up to date. A complete medical history, or even the routine diving medical history and examination forms, was not usually available

A statistical axiom is that absence of evidence is not evidence of absence. Questioning dead divers is not productive. Survivors, completing questionnaires, probably supply more accurate assessments. To avoid controversy, in this survey no attempt has been made to draw conclusions regarding the correlation between past illnesses and subsequent drowning. Table 3 shows the incidence of some disorders

Environmental factors

WATER CONDITIONS

The adverse influences of water conditions were as expected. Surprisingly over half the drownings occurred in

TABLE 3

Medical disorder	Fatalities*	Survivors
Asthma	10%	19%
Cardiovascular	6%	2%
Drugs	10%	8%
Very unfit	5%	4%
Panic	7%	8%

* History often not questioned

calm water as did 60% of the survivals. In 4% of the deaths calm conditions deteriorated. Moderately rough seas were associated with 25% of deaths and 40% of the survivals. Very rough conditions, hardly ideal diving conditions, were associated with 15% of the deaths. Currents were associated with 46% of the deaths and 31% of the survivals.

Strong tidal currents were more frequent (9%) in the fatality group than in the survivors (0%).

FRESH OR SEA WATER.

Most of the accidents occurred in the ocean without obvious differences between the fatality (93%) and survivor (98%) groups. The extra difficulty of performing rescues in cave diving (2% of the deaths) is obvious.

DEPTH OF INCIDENT

As in previous surveys,[1,3] over half the problems in the fatality cases were observed on the surface, although this was frequently related to the fact that the diver no longer had an adequate air supply to remain under water.

In referring to depth, we are measuring the commencement of the aspiration/drowning incident, not necessarily the original problem. For instance a diver who had been diving excessively deep (related to their experience), had used most of the air supply and then panicked and ascended, might then not show any evidence of aspiration until he reached the surface.

Approximately half the fatalities occurred while on the surface or on the way to the surface. Another 20% occurred in the first 9 m and the rest were distributed as shown in Table 4. This implies that just reaching the surface is not enough. Successful rescue requires the victim to remain there.

The survivors, probably because of the ability to supply a more detailed and specific history, indicated a greater incident depth. They probably more accurately represented the depth at which the incident developed, as opposed to the depth at which the incident was noted by others. Nevertheless, almost two thirds occurred in the top 10 m.

In the fatality and the survivor groups, the dive was the deepest of their diving career in 26% and 33% respectively. In almost half the "inexperienced" and "novice" divers the depth was beyond any previous dive, suggesting that these groups are especially susceptible to the various problems associated with depth (air consumption, poor visibility, narcosis, panic and logistic difficulty with rescue). This demonstrates that it is not so much the environment that is the problem, but the diver's experience of that environment. The danger of "diving deeper" without extra prudence and supervision is apparent. Any dive deeper than that previously experienced should be classified and treated as a "deep dive", irrespective of the actual depth.

TABLE 4

DEPTH OF INCIDENT

Incident depth	Fatalities	Survivors
Surface	51%	15%
During ascent	-	17%
1- 9 m	20%	33%
10-18 m	10%	15%
19-30 m	10%	13%
31-45 m	3%	6%
46-60 m	6%	2%
Diver's deepest dive	26%	33%

VISIBILITY

Visibility was usually acceptable, but seemed to be more frequently adverse in the fatalities (poor 26%, deteriorated 4% and night 5%) compared with the survivors (poor 18%).

ADVERSE ENVIRONMENTS

The cases, in general, demonstrated the adverse effects of various environments, especially with tidal currents (deaths 55%, survivals 31%), white (rough) water (deaths 44%, survivals 41%), poor visibility (deaths 26%, survivals 18%), cold (deaths 14%, survivals 12%) and deeper diving than previously experienced (deaths 26%, survivals 33%). There was not a great deal of difference between the two groups, except in the higher incidence of strong tidal currents, night diving and cave diving in the fatalities. The numbers, however, were small.

If such observations are valid, then they may reflect

either the effect on the victim or the problems with rescue produced by such environments.

Equipment

In most fatalities the equipment showed no structural abnormality. Only in 20% did significant or serious *faults* contribute to the fatality. This corresponded with the reported incidence by the survivors (18%). Minor faults observed probably would not have contributed significantly to the deaths (11%). Equipment faults were most frequently found with buoyancy compensators and regulators (both first and second stages).

Equipment *misuse* was more frequent but more difficult to ascertain in the fatality series and depends on one's definition (deaths 43%, survivals 38%).

Our definition of equipment misuse included the use of excessive weights (deaths 25%, survivals 27%) or the failure to carry equipment that could have been instrumental in survival such as buoyancy compensator, contents gauge, snorkel, etc. (deaths 12%, survivals 8%). Difficulties in using buoyancy compensators were frequent.

The incidence of equipment misuse would be much higher if one included a compromised air supply. Of equal interest was the failure to utilise equipment to ensure buoyancy following the incident. These are dealt with under diving technique.

Diving technique

Various diving techniques seemed to contribute to the drowning incidents, or influenced rescue and survival. They include a compromised air supply, buoyancy factors, buddy rescue and resuscitation attempts.

AIR SUPPLY

In 60% of the fatalities either an out-of-air (OOA 49%) or low-on-air (LOA 11%) situation had developed. There was insufficient air in the tank for either continuing the dive or returning to safety underwater.

In the survivors there were fewer incidents of compromised air supply (OOA 27%, LOA 8%), but it was still very frequent. The survivors tended to be more likely to have enough air in their tanks (more than 1/4 contents) to cope with an emergency (deaths 40%, survivals 65%). The difference is more if the cut off point is taken as half a tank (deaths 29%, survivals 45%).

In both groups failure to use the available contents gauge properly was common. This could sometimes be attributed to the conditions placing other stresses on the diver (depth, anxiety, tidal current, deepest dive ever, etc.). In many more cases there appeared to be a voluntary decision to dive until the tank was near reserve or "ran out".

One surprising feature was the failure in both groups (deaths 8%, survivals 13%) to turn on the tap of the scuba tank. Even though there was plenty of air in the tank it was unavailable, other than to sometimes allow a rapid descent to 10 m or so. Only then was the diver aware that further air was not available. In none of these cases had the diver breathed from the regulator before getting in the water nor had there been a buddy check of equipment nor an equipment check before descent.

In a smaller number of cases the diver had failed to turn the tap back on adequately, after checking the tank pressure and turning it off. The result was a partial restriction of the air supply, which became obvious only later in the dive or at depth.

BUOYANCY FACTORS

For survivors buoyancy was frequently a vital factor in reaching the surface and in remaining there as an unconscious diver. This gave the chance of being found, rescued and resuscitated in time. The 3 major influences on buoyancy are the buoyancy compensator (BC), weights and the companion (buddy) diver practice.

Buoyancy compensators

Failure to inflate the buoyancy compensator was common in both groups (deaths 52%, survivals 32%). In a few cases the buoyancy compensator failed to inflate for mechanical reasons (deaths 5%, survivals 8%). In 12% of the deaths the buoyancy compensator had been inflated before the incident.

In the survivor group the BC was inflated by the victim (deaths 15%, survivals 35%) or rescuer (deaths 16%, survivals 25%) in twice as many cases as in the fatality group (deaths 31%, survivals 60%). This figure is even more relevant when the delay in producing buoyancy in the fatality group is considered (see later).

Weights

In the vast majority of cases in both groups the victim did not drop the weight belt (deaths 86%, survivals 74%). Only about a fifth of both groups actually dropped their weight belts (deaths 13%, survivals 21%). Some unfortunates dropped their belts but became entangled (deaths 3%, survivals 2%). About the same number were not wearing weight belts (deaths 1%, survivals 6%).

Although in 30% of the fatality cases the weights were ditched, in practice this was not as valuable as it sounds. In most of the instances in which the rescuer ditched the weights (20), the victim was probably no longer salvageable, because of the delay (see later).

The survivor group not only ditched the weights more frequently, but more often it was done by the victims themselves, and when it was done by the rescuer, it was usually performed early in the incident.

Buoyancy action by victim

It appears that the fatality and survivor groups differed in that the latter tended to perform an action, either inflating the BC (deaths 15%, survivals 35%) or successfully dropping the weight belt (deaths 10%, survivals 19%), which resulted in them achieving positive buoyancy during and following the incident.

An interesting observation was that when the victim and buddy were both in difficulty, usually because of a LOA/OOA situation, irrespective of whose problem developed first, the overweighted diver tended to be the one that died and the buoyant diver the one that survived. The ratio of the two were 6 to 1, in the 14 instances.

All this gives support to the current emphasis by Instructor agencies on buoyancy training, although one could argue for its inclusion in the introductory courses more than the advanced courses. However, there is obviously a need to emphasise that weight belts should be dropped when in trouble.

COMPANION DIVER PRACTICE, RESCUE AND RESUSCITATION

In most cases of significant aspiration of water, rescue depends on rapid action being undertaken by either the victim or the companion (buddy) diver. Once a diver gets into difficulty and is unable to carry out safety actions by himself, he is heavily reliant upon his buddy or dive leader.

The fatality and survivor populations were so different in this respect, that we had to separate them.

FATALITIES

Buddy experience

In the fatality group only 41% of the victims had an experienced buddy. Twenty one percent were diving alone and 32% were diving with a novice, or less experienced, diver. In the majority of cases the buddy's experience hardly mattered as only in 8% of the deaths was a buddy present throughout the incident.

Buddy diver/group practice

In 21% of the fatalities, the dive was a deliberate solo one. Voluntary separation had occurred in 50% of the deaths before the victim died. In 38% the diver had separated from his buddy, and in another 12% from the group, before the incident. Separation was initiated in 31% because the victim could not continue the dive (usually due to a LOA situation). The victim then attempted to return to the surface alone, a solo dive, so that 52% of the fatalities were alone when they died.

The diver was separated from his buddy or the group during the actual incident, and often by the incident, in 21% of cases. But, in almost half of these cases the separation was because the diver was behind his buddy or the group. The others occurred during the "rescue".

The diving practice of voluntarily separation in 71% of cases made early rescue and resuscitation improbable. Another 9% were swimming behind their companion/s and the victim was not visible to the "buddy" at the time of the incident. In fact, 80% of the victims did not have a genuine buddy, by virtue of their elected diving practice.

In less than one in ten deaths (8%) was there a continued contact with the buddy or group during and following the incident.

The fatalities seemed to have flagrantly disregarded the "buddy" system, as did their companions, the organisation which conducted the dive, or the "dive leader". Group diving conferred little value because the "leader" often had insufficient contact with individual divers to be classified as a buddy and the responsibility of others was not clear, especially towards the last of the "followers".

Rescuer action

As the buddy system was essentially not used in the fatality group, the commonest response to the accident by the other divers was that no attempt was made to rescue the victim (31%). The second commonest response was that an attempt was made, but failed (24%). This is understandable, when one appreciates that no one knew where the victim actually was. A smaller number of rescuers actually found the victim and attempted a rescue, with some initial response by the victim (17%).

In a quarter of the cases there was no search for the victim until after the planned dive had been completed and it was realised that the victim had not returned. In these cases there was a search for the body, which failed in most

cases. In a number of the cases it was not the original buddy or group diver who undertook the attempted rescue or search. Sometimes it was other divers who were coincidentally in the same area. In other instances coincidental observers, from shore or boat, were aware of the victim's distress and undertook the rescue attempt.

Body search

There was no need for a body search in 26% of the deaths. A formal search for the diver's body, undertaken separately and usually well after the dive, was successful in a third of the cases, with another third successful during future attempts. In a very small number (7%) the body was found coincidentally and in only 3% was it never found.

Resuscitation

Resuscitation was not a feasible option for most of these cases, who were obviously dead or showed no response to the rescuers attempts in 9 out of 10 cases. This is explained by the excessive delay in the rescue in most cases. There was an initial response to resuscitation in 7% and ineffectual resuscitation was applied to 2%.

Delay in rescue

In only 20% was the diver rescued within 5 minutes of the probable incident time, giving a real chance of successful resuscitation. In another 12% the diver was recovered within 5-15 minutes. Theoretically there was a slight chance of recovery for these divers, had the rescue facilities been ideal and had fortune smiled brightly. Recovery of the body took 16-60 minutes for 19%, hours for 36%, days for 5%, weeks for 5% and 3% of the bodies were never found.

Autopsy

In 10% either the body was not available or an adequate autopsy was not performed. In the remaining 90% the autopsies were either routine (72%) or conducted specifically by a diving pathologist (18%).

SURVIVORS

In the surviving group most were rescued by their companion. Some form of artificial respiration or CPR was required in 29% of the cases. Oxygen was available and used, usually in a free-flow system, in 52% of cases.

Rescuers behaviour

No specific data is available on the buddy divers assisting the survivors, other than the subjective assessment as to whether the survivor believed the buddy to be of much value. The buddy was immediately available to the survivor in 71%, was considered to be of assistance in 58%, supplied an independent air source in 15%, inflated the BC in 25%, ditched the weight belt in 25% and attempted buddy breathing in 4%. In 52% the diver surfaced under the buddy's control.

The attitude toward buddy diving practice with the survival group appeared to be very different to those in the fatality group. To successfully rescue an incapacitated diver one must know where he is and reach him quickly. This implies some buddy responsibility. The buddy divers in this series seemed to be of considerable value once they reached the victim, implying good training in this aspect of diver safety. The high figure for oxygen utilisation must represent a more sophisticated and organised diving activity, which may also be related to a more conscientious attention to responsible buddy behaviour.

Of recent years there has been a promotion of solo diving and reliance upon oneself rather than on a buddy.

Denying or repudiating the hard-learnt lessons of the past is fashionable and innovative, implying a diving expertise and an avant garde approach, as well as ensuring an audience. In diving medicine it is also easier than acquiring practical experience or doing the hard data collection. In this instance, as in others, these "experts" may well be misleading both their contemporaries and the diving trainees.

Conclusions

There are many lessons to relearn from this survey, as well as from the diving medical experience of the past, to reduce the likelihood of drowning with scuba (Table 5). Drowning prevention measures fall into three groups, Before and during the dive (1-7), When a problem develops (8 and 9) and survival requirements (10). They can be summarised as follows.

1 Personal factors

Ensure both medical and physical fitness, so that there is no increased likelihood of physical impairment or loss of consciousness or difficulty in handling unexpected environmental stresses.

2 Experience

Ensure adequate experience of the likely dive conditions (dive under the supervision of a more experience diver, when extending your dive profile).

TABLE 5

DROWNING PREVENTION

Before and during dive

1 Personal fitness
2 Experience adequate for the dive
3 Equipment checked and working properly
4 Environment safe for experience level
5 Buoyancy (dive neutral)
6 Air supply adequate, turned on and monitored
7 Buddy diving done properly

When a problem develops

8 Increase buoyancy (become positive)
Unbuckle weights and inflate BC
9 Inform buddy, if feasible

Survival depends on

10 Rescue, first aid and medivac
All need to be planned before the dive

3 Equipment

Although faults with diving equipment are inevitable, they are a less frequent contributor to drowning than misuse of equipment. The latter includes the practice of overweighting the diver and over reliance upon the buoyancy compensator. Failure to possess appropriate equipment is a danger, but not as much as the failure to use the equipment safely. Permitting a compromised air supply is dangerous.

4 Environment

Hazardous diving conditions should be avoided. Use extreme caution with tidal currents, rough water, poor visibility, enclosed areas and excessive depths.

5 Neutral buoyancy

Ensure neutral buoyancy while diving. This implies not being overweighted and so not being too dependent on the buoyancy compensator.

6 Air supply

An inadequate supply of air for unexpected demands and emergencies may convert a problematical situation into a dangerous one. It also forces the diver to experience surface situations that are worrying and conducive to anxiety, fatigue, unpleasant decision making and salt water aspiration.

Equipment failure is not as common a cause of LOA/OOA as failure to use the contents gauge and/or a decision to breath the tank down to near reserve pressure.

7 Buddy diving.

Use traditional buddy diving practice, 2 divers swimming together. Solo diving, for the whole or part of the dive, is much more likely to result in an unsatisfactory outcome in the event of diving problems. It is the divers who are committed to the traditional buddy diving practices who are likely to survive the more serious of the drowning syndromes.

8 Become positively buoyant.

Positive buoyancy is usually required if problems develop. This can be done by dropping weights and inflating the BC.

Failure to remove the weight belt during a diving incident continues to be the major omission and must reflect on training standards. In most situations, unbuckling and then ditching (if necessary) the weight belt is the most reliable course of action once a problem becomes evident. Buoyancy compensators cause problems in some emergency situations and, not infrequently, will fail to provide the buoyancy required. They are of great value in many cases, but are not to be relied on.

9 Buddy communication

If feasible, inform the buddy prior to ascent. If correct buddy diving practice is being carried out, he will automatically accompany the injured or vulnerable diver.

10 Rescue

Rescue, first aid and medivac need to be planned before the dive. Employ the rescue, water retrievals, first aid facilities (including oxygen) that have been discussed before the dive. Know how to contact the medivac systems and have the necessary equipment before the dive.

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*Its companion paper (*Drowning syndromes: the mechanism*) will appear in the March 1998 issue of the SPUMS Journal.*

THE WORLD AS IT IS

AUSTRALIA'S UNDERWATER LINE DANCING RECORD ATTEMPT

Peter Fields

Following the spectacular growth and success of the Historical Diving Society in the USA, an outfit which has attracted diving luminaries such as Hans and Lotte Hass, Jean-Michel Cousteau, E R Cross, Bev Morgan, Phil Nuytten, Dr Sylvia Earle and many other celebrities too numerous to mention, our own representative Bob Ramsay, the expatriate Scot from Adelaide, set up a branch in 1996 in Australia. Since then the Diving Historical Society Australia SE Asia has blossomed and is attracting people from all parts. Folk with a love of diving and its history, collectors and restorers of old equipment and aficionados of standard dress (hard hat) diving.

After a succession of social get-togethers in the region Bob was looking for ideas for something a bit more grand. Over a few beers one Adelaide evening Bob was musing on what to do with, say, 3 or 4 helmet divers in a pool. John Riley, in a flash of alcoholinspired mischief, suggested a line dancing competition. Bob was entranced and passed on the idea to Melbourne's John Allen. So was born the Word Record Attempt for Underwater Line Dancing.

John Allen ran with the idea and, using his brilliant organising skills, created not only a superb record attempt but a whole weekend of entertainment and recreation for standard dress lovers, society members and their friends from all over Australia.

On a fine spring Saturday in October nine men and one woman in traditional helmet diving dress, plus all the myriad paraphernalia which accompanies this activity, lined up at the Harold Hold Swim Centre in Glen Iris (in suburban Melbourne), Victoria, ready to create a record.

John Allen had planned the event down to the last detail; pallets of 240 cu ft cylinders, delivered by BOC Gases, were on hand, media fact sheets had been handed out to print and electronic media, a detailed scheme of underwater arrangements was set out on a white board, a cook tent and canteen set up to provide breakfast and lunch for the participants and a local bootscootin' club alerted to be on hand to coach the tyro standard-dress line dancers.

In addition, topside and underwater supervisors were briefed and ready to direct and assist the divers, easy access to the 5 metre deep pool was provided via builders' ladders and a range of trophies and certificates awaited the successful conclusion of the dive. Awards were to be given for: Best Presented Equipment, Best Dressed Dive Crew and, most coveted of all, Best Underwater Line Dancer.

To further illustrate the depth and detail of John's organising skills, extra dive crew hands were on duty to assist, spare Desco cuffs were on stand-by in case of blow-out and Melbourne dive historian Jeff Maynard and your correspondent were detailed to handle media arrangements and enquiries.

Fig 1. The venue

By 0900 the poolside was a buzz of activity. Sydney's contingent had a crew of 4 divers and support including a diver all the way from Cairns in North Queensland. All were impeccably attired in matching shorts and gold t-shirts topped with matching Akubra slouch hats. Missing, however, were matching Oroton handbags. Graham Weir had arranged a rental truck and had ferried all the heavy equipment many kilometres down the track from Sydney. Included in the Sydney equipment was a beautiful handcrafted wooden diver-distribution and communications panel, a real work of art.

Melbourne, the home team, had a fine line-up of divers including Mandy Jones, the only lady entrant. Frank Zeigler had trucked over his gear from Portland in West Victoria and "Wee Bobby" Ramsay transported his equipment all the way from Adelaide, South Australia.

By 1000 the joint was jumping. You could not get near the divers for media crews, Betacam cameras and foxtail mikes on long booms. The media loved it. All the ingredients were there: colour, movement, novelty, history, the romance of diving and, to cap it all, the uniqueness of underwater line dancing.

Craig Jones, topside dive supervisor, briefed the divers on positions, procedures and safety precautions while Geoff Cross prepared for his in-water dive supervisor role. Underwater cameramen readied their equipment, both still and video, Joy Allen the resident chef, cooked resolutely on and Stampede Line Dancing's Cherine Stiller and her team taught the guys and gal their basic underwater dance steps.

The dive, as a result of the impeccable topside organisation, went like clockwork. The divers finished dressing and in an orderly fashion took up their designated positions, in two lines of 5, underwater, making sure all comms were operative and no hoses or lines were snagged. All ready, the music and dance commenced. Ben Cropley, bless his soul, boogied his heart out to emerge clear winner of the Best Underwater Line Dancer trophy.

Cameras flashed and video cameras whirred and the team clumped through their routine. As the dance ended Ben, to amuse the folk topside, did a few balloon ascents, popping his rig out the water clear to his knees, most spectacular.

That was not the end of the weekend. The rest of the day was spent with support crews and others diving the equipment, fun and sport with re-breathers, modern Superlite helmets and general hoop-de-do in the pool, before cleaning up. A great dinner party and presentation was held at Chez Lucien in Moonee Ponds on Saturday evening. Incidentally the previous night, Friday, provided the opportunity for a get-together, drinks and a meal at a grand old pub in South Melbourne. This convivial meeting set the tone for what was to be, in every sense, a great weekend of fun and entertainment.

The Melbourne lads had also organised the Polly Woodside (a fine old sailing ship moored in Melbourne's docklands) Maritime Museum as avenue for a talk fest on Sunday morning. Jeff Maynard gave a fine presentation which included footage from early Australian movies of the

Fig 2. The well dressed lady diver

Fig 3. Essential equipment for underwater line dancing

1930's which featured standard dress divers and all sorts of derring-do. Bob Wallace-Mitchell reminisced about the early diving days with Ted Eldred and the development of the Porpoise set and the growth of diving as a sport. A wartime Commonwealth Marine Salvage Board diver, Kevin Hood, entertained with stories of post Japanese air-raid salvage work in Darwin, salvage in the Pacific war theatre and of his time with the legendary Captain Williams (of Niagara gold recovery fame) and diver Johnno Johnstone.

For lovers of old equipment the divers line-up was:

Mandy Jones	Modified MK5 US Navy, 1944 vintage.
John Allen	1970's Yokohama with large face plate (probably fitted for California abalone diving).
Link Kirby	Seibe-Gorman
Ben Cropley	Seibe Gorman, 4-light construction helmet
Bob Ramsay	1943 MK5 Desco
Darren Skerman	US Navy MK5 Schrader
John Balson	Shanghai Diving Work 12 bolt (Chinese)
Michael Graves	Russian Navy 3 bolt
Peter Weir	Toa 12 bolt (Japanese)
Graham Weir	Siebe Gorman 6 bolt.

Contacts

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Who Was Who On The Day:

Craig Jones	Topside Dive Supervisor
Geoff Cross	In Water Helmet Dive Supervisor
Jeff Maynard	Media Liaison
Peter Fields	Media Liaison
Pat Forbes	In Water Media Liaison
Narelle Wilson	Topside Media Supervisor/Assistance
Peter Bathie	Video Cameraman
Laurie Bell	Video Cameraman
Joy Allen	Head Chef

Awards

Best Presented Equipment	Sydney Crew
Most Original Equipment	Bob Ramsay, Adelaide
Best Underwater Line Dancer	Ben Cropley, Melbourne
Best Dressed Crew	Sydney Team

SPUMS NOTICES

THE SPUMS POLICY ON THE INITIAL MANAGEMENT OF DIVING INJURIES AND ILLNESSES

Des Gorman, Drew Richardson,
Mike Davis, Richard Moon
and James Francis

Key Words

Accidents, decompression illness, first aid, injuries, policies, rescue, transport, treatment

An introduction to SPUMS policies

The Society considers education and dissemination of information to be among its primary roles. This is the rationale for a Society Journal. The Society is also often asked for opinions on subjects in diving practice and health. This is the rationale for Society policies. On occasions, these policies have been the product of individuals or small working groups. More recently, workshops have been used to produce substantial policies (e.g. emergency ascent training,[1,2] computer-assisted diving,[3] asthma,[4] certification of diving fitness[5] and recreational technical diving[6]). In future, the newly formed Ex-Presidents Committee will be asked to develop some Society policies.

A SPUMS policy is intended to be a statement of best practice. While such policies are based on the concept of practicability, they are not intended to be drafts for subsequent codes or regulations. The concept of practicability deserves some explanation. In the context of Society policy, this refers to something being achievable. Practicability is not synonymous with convenient. For example, in an ideal world, all injured and ill divers would be rescued from the sea in an horizontal position and all would undergo intravenous fluid resuscitation. Are these responses practicable ? An horizontal rescue is achievable already in the majority of diving situations, given some forethought and practice, and achievable for most others given minor modification. Consequently, an horizontal rescue is considered to be practicable and is cited in the Society policy. Conversely, it is almost impossible to maintain intravenous infusion skills for existing para-medics, let alone recreational dive instructors, dive masters and charter boat operators. Consequently, with the exception of diver medical technicians, a requirement for anyone supporting diving to be able to undertake intravenous fluid resuscitation is considered impracticable and is not cited in the Society policy.

SPUMS policy on the initial management of diving injuries and illnesses

Introduction

There is a plethora of diving first aid protocols and procedures available to the diving and medical communities. Some of these are in conflict and the Society has been asked to clarify its position in this context. In addition, there are data that now support an increasing role for some drugs in the treatment of decompression illness (DCI). The potential for the latter to be widely recommended as first aid measures needs to be determined and included in any policy on the initial management of injured and ill divers.

Because of the differing interpretations of the term “first aid”, this policy is deliberately titled, *The initial management of diving injuries and illnesses.* This policy describes a generic approach to the initial management of an unwell diver, regardless of the nature of the injury or illness. This is in recognition of the difficulties that most medical practitioners and essentially all divers have with specific diagnoses. The policy aims to describe initial management that does not require an accurate diagnosis and is divided into the following sections:

1 training requirements;
2 resuscitation equipment and supplies;
3 rescue and resuscitation;
4 posture;
5 oxygen administration;
6 fluid resuscitation;
7 drug therapy and pain relief;
8 communications systems and retrieval;
9 management of specific conditions.

The term occupational diver here is used in the modern context of any diver who dives for pay or reward. The term commercial diver is used to distinguish all other occupational divers from recreational dive instructors.

Training requirements

All occupational divers should be trained in basic life-support techniques. There is a need for occupational divers to be able to demonstrate an ongoing (annual) competence in airway management, expired air resuscitation (EAR), external cardiac compression and administration of oxygen, at as close as is possible to 100% oxygen, to both conscious and unconscious people. Recreational divers should be encouraged to acquire and

maintain these skills by undertaking advanced diver training modules and/or by attending first aid courses run by organisations such as St John Ambulance. These recommendations are consistent with the policy goals of the Australian and New Zealand Resuscitation Councils.

While the ability to administer intravenous fluids is highly desirable, access to training in this is limited. Only diver medical technicians should be expected to demonstrate an ongoing competence in intravenous line insertion and fluid administration.

Resuscitation equipment and supplies

All dive platforms should have a proven and exercised system to rescue an unconscious diver from the water horizontally. Similarly, shore based operations should have a proven and exercised procedure for removing a diver from the water horizontally.

All commercial dive and dive-training platforms should comply with local regulations and codes with respect to the provision of an on-site diver medical technician and should also have equipment and supplies immediately available to enable:

10 oxygen administration (see Note 1);
11 intravenous fluid administration;
12 compliance with the local diving codes and or regulations.

All platforms and shore based operations used to train recreational divers and all vessels that are chartered to recreational divers should also have a first aid kit (an example of a suitable inventory is that developed by PADI and published in their Rescue Diver Manual[7]) and equipment and supplies available to enable oxygen administration (see Note 1). It is also recommended that, where possible, this level of equipment and supplies be available at all dive sites.

NOTE 1.
With respect to oxygen administration, the following comments apply:

13 oxygen should be administered at as close as is possible to 100% oxygen to both conscious and unconscious people;
14 the volume of oxygen needed for any diving operation should be enough to supply the ventilatory needs of two divers simultaneously throughout a retrieval to the nearest facility with oxygen supplies;
15 more oxygen will be needed for a demand flow system than for a rebreather circuit and even more oxygen is needed for a continuous flow apparatus.

Rescue and resuscitation

Recovery of a free-swimming diver from underwater should follow the techniques currently taught by the recreational dive instructor agencies and the licensed occupational diver training schools. The recovery to and resuscitation of a diver on or in a stage, open or closed bell will be determined by the specific diving system in use.

Expired air resuscitation in the water should never delay the recovery of a diver to a diving platform or ashore. There are sufficient doubts about the safety and efficacy of EAR in the water at present (April 1997) to prevent the general recommendation of this technique.

Resuscitation of a diver should be conducted in accordance with the current guidelines of the Australian Resuscitation Council or equivalent national organisation.

The re-warming of a cold diver should be based on the following.

16 The avoidance of any further cooling by removing wet clothing (with the possible exception of wet suits) and insulating the diver with warm, wind-proof material(s). The head should be covered.
17 If intravenous fluids are administered, these should be warmed, but to no more than 45°C. Oral fluids should also be warmed, but should only be given to fully conscious persons.
18 A cold diver who has stopped shivering or who is unconscious represents a medical emergency and requires urgent evacuation to the nearest appropriate treatment facility.

Posture

The best posture for most injured divers (the exception being those with isolated vertigo and nausea, with or without hearing loss) is horizontal. This may be impossible in diving bells because of the limited available space. The recovery position (as defined and taught by organisations such as St John Ambulance[8]) should be used for patients who are unconscious or drowsy or where there is some other concern for their airway (e.g. fractured jaw).

Divers with vertigo should be encouraged to keep still and not to do anything which could cause additional stress to the inner ear, such as a Valsalva manoeuvre or straining.

Sitting or standing up a diver to perform tests of balance and gait, or to undertake investigations such as a chest X-ray, should only be undertaken by a physician and then only after specific contraindications are excluded (such as a significant risk of cerebral arterial gas embolism, postural hypotension and severe vertigo). Preferably, this

should be delayed until the patient arrives at a definitive treatment facility, one which has a recompression chamber (RCC).

Administration of oxygen

Oxygen should be stored and used in accordance with local standards, codes or regulations.

Oxygen should be administered to all injured and ill divers at as close as is possible to 100% oxygen. With the specific exception of instructions from a physician at a facility which has agreed to accept an injured or ill diver for hyperbaric treatment, this administration of oxygen should not be interrupted.

There are sufficient doubts about the safety and efficacy of oxygen therapy in the water to currently prevent the general recommendation of this technique.

Fluid resuscitation

Intravenous administration is the preferred method of fluid resuscitation for any severely injured or ill diver. This should either be conducted according to a fixed protocol (such as an initial regimen based on one litre of normal saline given as fast as is possible, followed by alternating a litre of Hartmann's solution and normal saline over 4 hours) or adjusted according to clinical parameters (such as urinary output and/or haematocrit on a physician's instructions). Glucose containing intravenous fluids should not be given to an injured or ill diver, although an appropriate amount of glucose in an oral fluid (80 to 120 Mm, such as 20 g per litre) will enhance the rate of water absorption without causing an appreciable increase in plasma glucose.[9]

Suitable oral fluids[9] can be given to injured or ill divers, but only under the following circumstances:

19 the diver is fully conscious, is not nauseated and there is no concern for the airway;
20 the diver has been walking around or sitting up before first aid was started;
21 the administration of oxygen will not be interrupted for more than a few minutes each time.

An accurate record of fluid administered and urinary output should be kept for any injured or ill diver. Bladder catheterisation by or on the order of a physician may be necessary.

Drug therapy and pain relief

There are at present (April 1997) no drugs that can be recommended for the initial management of diving injuries and illnesses. Inhalation of nitrous oxide (such as Entonox) for pain relief should never be used for analgesia in anyone suspected of decompression illness (DCI). Parenteral administration of analgesics should only ever be undertaken on the instructions of a physician.

Communications systems and retrievals

All dive operations should have systems in place to provide immediate contact with the local diving emergency services and the local emergency services to obtain advice about initial management, regional retrieval systems and treatment facilities. An accurate record of events should be kept.

An acutely injured or ill diver should be retrieved to the nearest suitable treatment facility by the most appropriate method possible, providing that the following conditions are recognised or apply:

22 any retrieval should be timely as it is likely that the longer the delay for a diver with DCI to recompression, the worse the outcome;
23 any necessary resuscitation, oxygen administration and fluid therapy should not be compromised by the retrieval;
24 if DCI is possible, then the retrieval should occur as close to sea level as is possible.

An altitude of 300 m is considered the maximum that should be allowed during the retrieval of a diver suspected of suffering from DCI. However, situations do arise where a commercial aircraft with a cabin pressure equivalent to an altitude of 2,400 m is the only option available. Clearly, this option is not ideal and should be accompanied by continuous oxygen administration.

A transportable RCC can be used to transport divers with DCI, but given the cost, logistical problems and dangers involved, such a RCC should only be employed under appropriate conditions and with sufficient support.[10]

Management of specific conditions

The management of marine animal envenomations should follow standard guidelines.[11] Management of omitted decompression, which includes procedures for the management of decompression illness, is shown in a flow chart on page 197. A number of diving accident first aid flow charts, which have been published in the SPUMS Journal over the years, appear on pages 198 and 199. A flow chart on page 200 covers diving accident procedures.

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Dr Michael Davis, FANZCA, Dip DHM, is Director of the Hyperbaric Medicine Unit at Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand. He was Convener of the 1997 Annual Scientific Meeting.

Professor D F Gorman FAFOM, PhD, is Associate Professor of Medicine and Head, Occupational Medicine, Faculty of Medicine and Health Sciences, University of Auckland, New Zealand. He is the immediate Past-President of SPUMS.

Dr T J R Francis, MSc, PhD, Dip DHM, one of the Guest Speakers at the 1997 Annual Scientific Meeting, was until 1996 Head of Undersea Medicine at the Institute of Naval Medicine, Alverstoke, Gosport, Hampshire PO12 2DL, England.

Professor Richard E Moon was one of the Guest Speakers at the 1997 Annual Scientific Meeting at Waitangi, New Zealand.

Drew Richardson is Vice-President, Training, Education and Memberships of PADI Worldwide and President, Diving Science and Technology, Inc.

CONSTITUTIONAL AMENDMENTS

The following motions amending the constitution were passed at the Annual General Meeting on Saturday 19/4/97 at the Quality Resort Waitangi, New Zealand.

1 That Rule 3 Life Members, (b) be altered by replacing the word *five* in the last sentence by the word *eight*.

The new sentence would read: *The number of life members shall at no time exceed eight nor shall more than one such member be elected in the one financial year.*

2 That Rule 23. (a) (ii) be changed by replacing *28* by *56*.

The new wording would be: *Shall be received by the Secretary of the Association not less than 56 days prior to the date of the annual general meeting*.

The amendments will not come into effect until approved by the general body of members. Any member who objects to any of the amendments should notify the Secretary of SPUMS, Dr Cathy Meehan, C/o Australian and New Zealand College of Anaesthetists, 630 St Kilda Road, Melbourne, Victoria 3004, Australia, in writing before February 1st 1998. If any member objects a postal ballot will be held. If no objection is received it will be assumed that the membership has voted in favour of the amendments.

Cathy Meehan
Secretary of SPUMS.

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY ANNUAL SCIENTIFIC MEETING

Palau May 8th-17th 1998

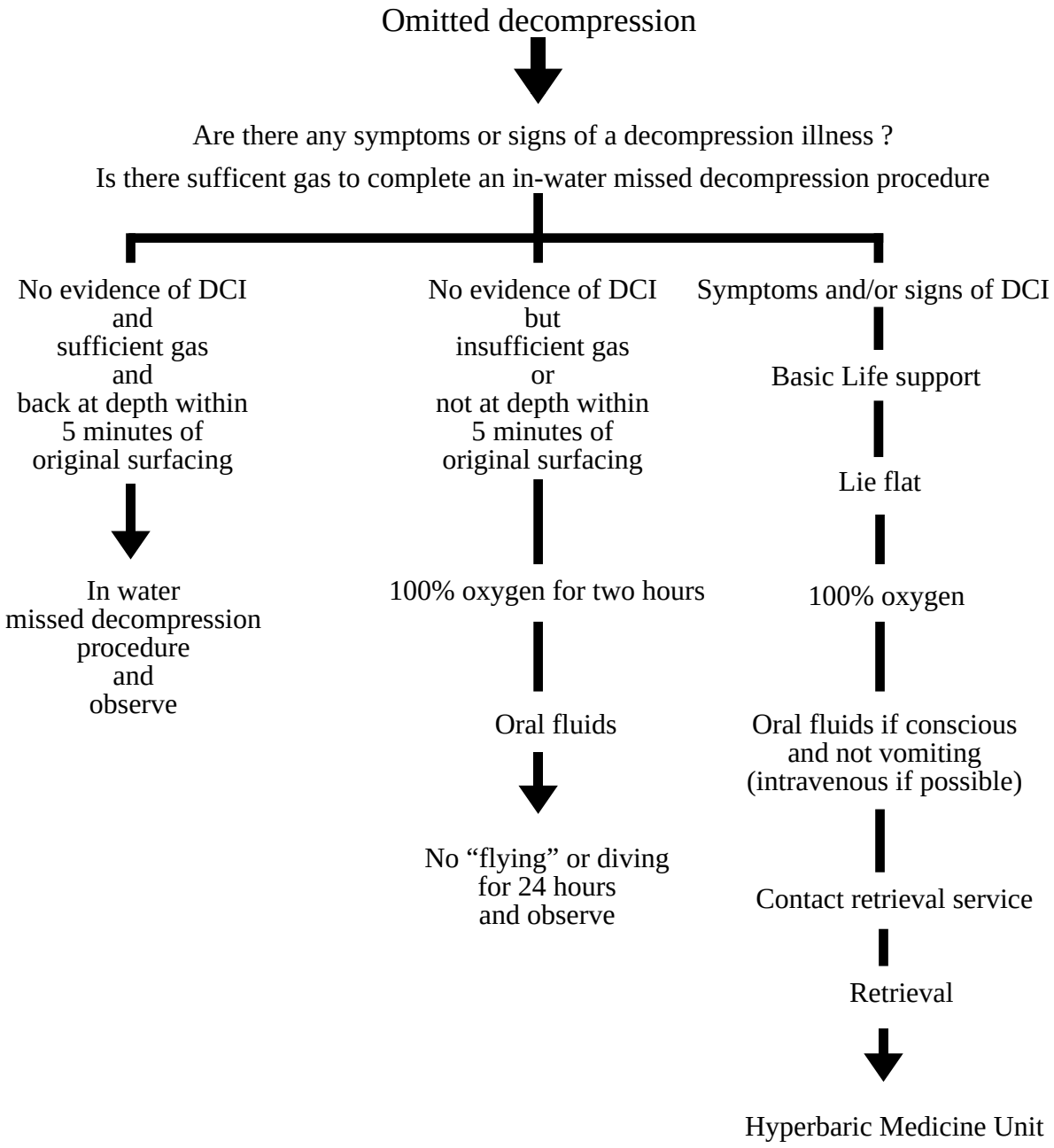
The Guest Speakers are Professor David Elliott (UK) and Dr John Bevan (UK). The Convener of the Annual Scientific Meeting is Dr Chris Acott. The theme of the meeting is *Highlights from the History of Diving and Diving Medicine*. The workshop theme is *The Ageing Diver*.

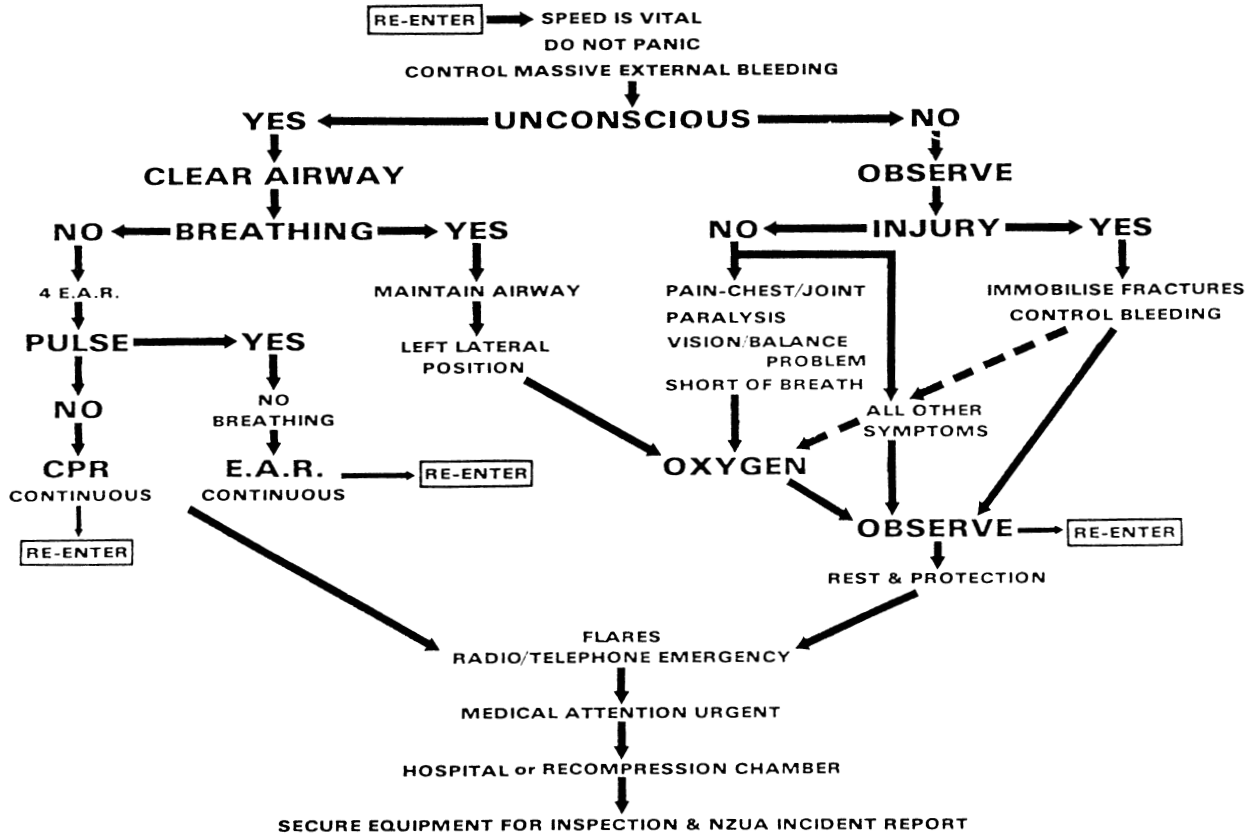
To present papers contact: **Dr Chris Acott**
Hyperbaric Medicine Unit, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000
Telephone +61-8-8222-5116. Fax +61-8-8232-4207.
E-mail guyw@surf.net.au

Speakers at the ASM must provide the printed text and the paper on disc to the Convener before speaking.

The Official Travel Agent for the meeting is:
Allways Dive Expeditions
168 High Street, Ashburton, Victoria, Australia 3147
Telephone 03-9885-8863. Toll Free 1-800-338-239
Fax 03-9885-1164.

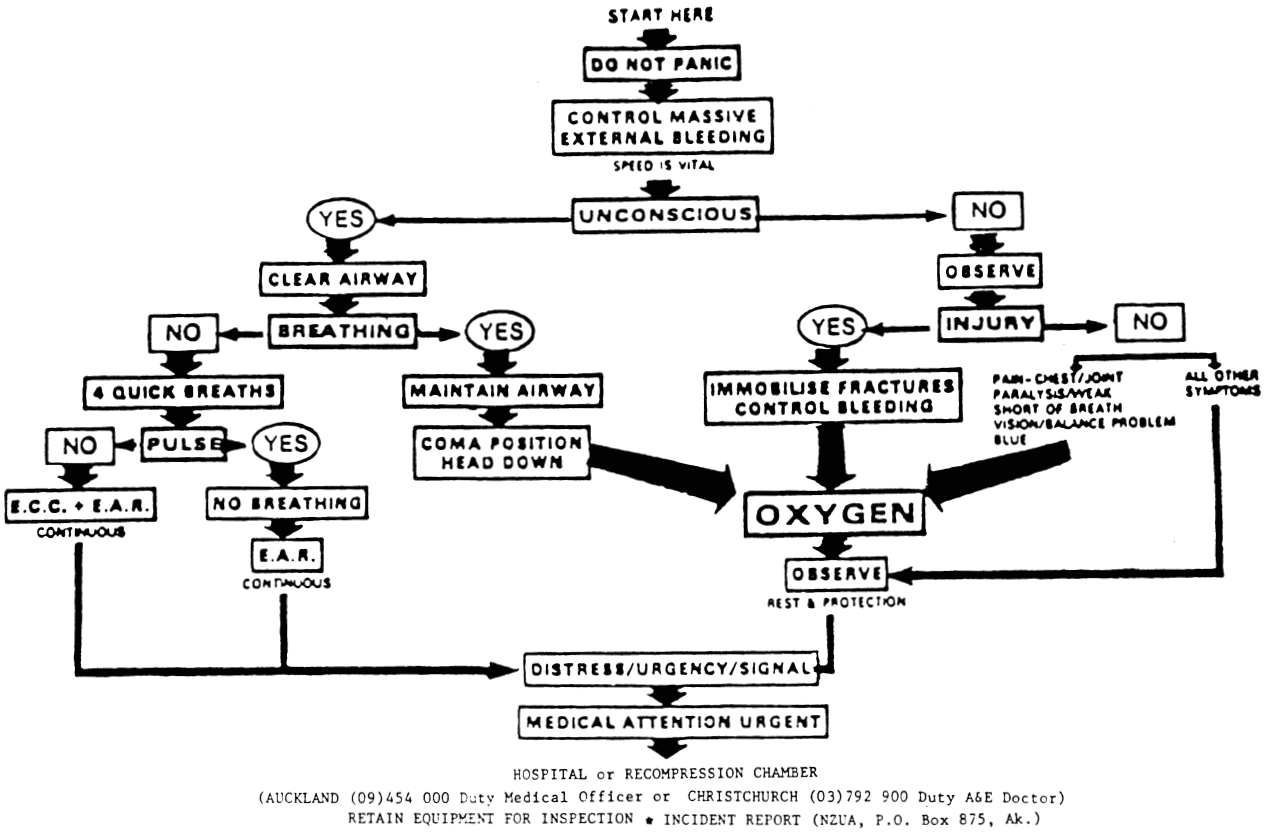
OMITTED DECOMPRESSION PROCEDURES



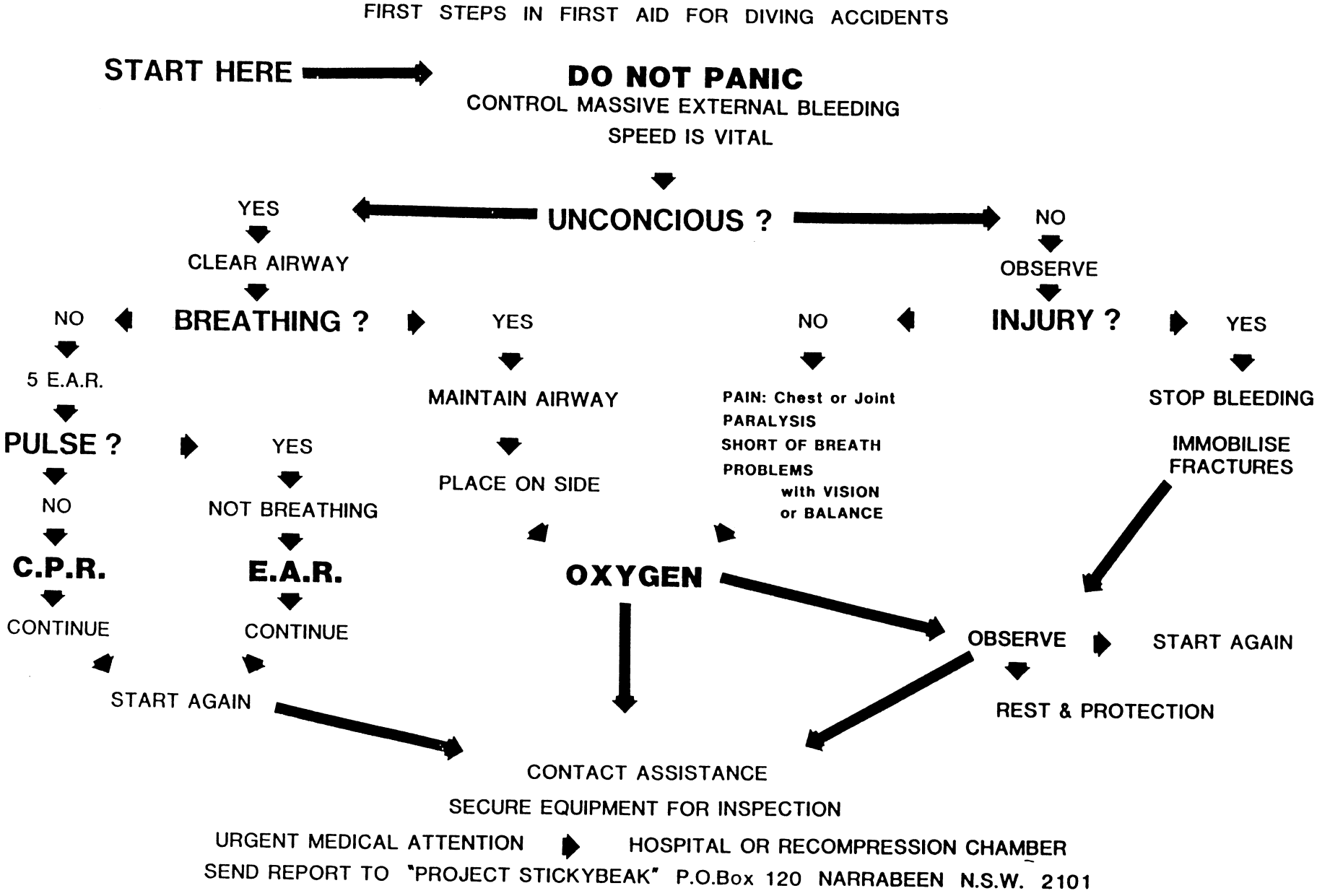


Diving First Aid decision tree for intial management 1980 version (Mike Davis)

On page 199 is John Knight's 1983 modification of Mike Davis' 1980 chart produced for Australian divers

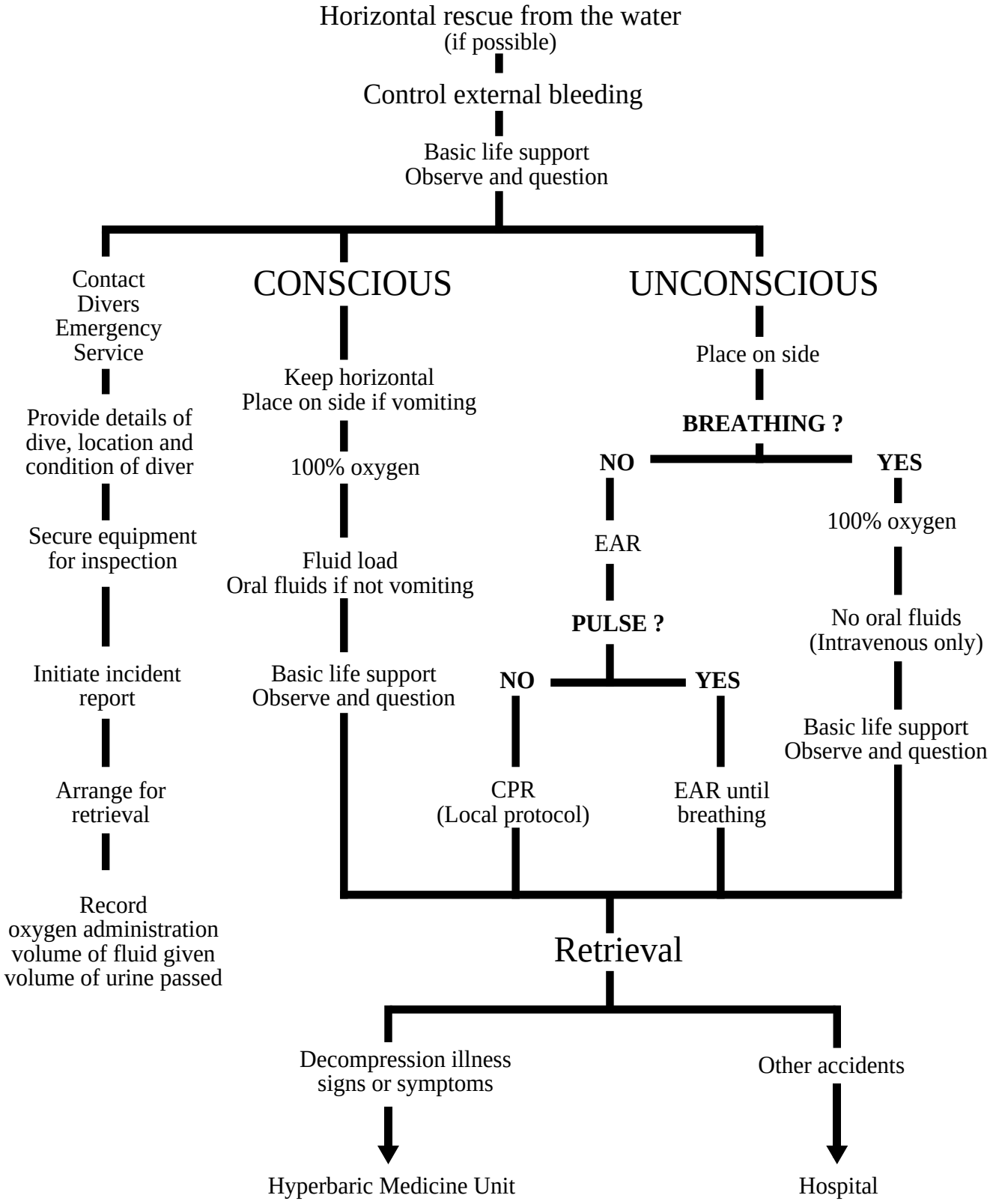


New Zealand Underwater Association First Aid for diving accidents Chart (1985) based on one by Mike Davis


FIRST STEPS IN FIRST AID FOR DIVING ACCIDENTS
START HERE
DO NOT PANIC
CONTROL MASSIVE EXTERNAL BLEEDING
SPEED IS VITAL
UNCONCIOUS ?
YES
CLEAR AIRWAY
NO
OBSERVE
BREATHING ?
NO
YES
INJURY ?
NO
YES
5 E.A.R.
MAINTAIN AIRWAY
PAIN: Chest or Joint
PARALYSIS
SHORT OF BREATH
PROBLEMS
with VISION
or BALANCE
STOP BLEEDING
IMMOBILISE
FRACTURES
PULSE ?
YES
PLACE ON SIDE
NO
NOT BREATHING
C.P.R.
E.A.R.
OXYGEN
CONTINUE
CONTINUE
OBSERVE
START AGAIN
START AGAIN
REST & PROTECTION
CONTACT ASSISTANCE
SECURE EQUIPMENT FOR INSPECTION
URGENT MEDICAL ATTENTION
HOSPITAL OR RECOMPRESSION CHAMBER
SEND REPORT TO "PROJECT STICKYBEAK" P.O.Box 120 NARRABEEN N.S.W. 2101

SPUMS

DIVING ACCIDENT PROCEDURES



SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE.

Requirements for candidates

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the Society, the candidate must comply with the following conditions:

1 The candidate must be a financial member of the Society.

2 The candidate must supply documentary evidence of satisfactory completion of examined courses in both Basic and Advanced Hyperbaric and Diving Medicine at an institution approved by the Board of Censors of the Society.

3 The candidate must have completed at least six months full time, or equivalent part time, training in an approved Hyperbaric Medicine Unit.

4 All candidates will be required to advise the Board of Censors of their intended candidacy and to discuss the proposed subject matter of their thesis.

5 Having received prior approval of the subject matter by the Board of Censors, the candidate must submit a thesis, treatise or paper, in a form suitable for publication, for consideration by the Board of Censors.

Candidates are advised that preference will be given to papers reporting original basic or clinical research work. All clinical research material must be accompanied by documentary evidence of approval by an appropriate Ethics Committee.

Case reports may be acceptable provided they are thoroughly documented, the subject is extensively researched and is then discussed in depth. Reports of a single case will be deemed insufficient.

Review articles may be acceptable only if the review is of the world literature, it is thoroughly analysed and discussed and the subject matter has not received a similar review in recent times.

6 All successful thesis material becomes the property of the Society to be published as it deems fit.

7 The Board of Censors reserves the right to modify any of these requirements from time to time.

MINUTES OF THE SPUMS COMMITTEE TELECONFERENCE

held on 14/9/97

Opened at 1010 Eastern Standard Time.

Present

Drs G Williams (President), C Meehan (Secretary), T Wong (Treasurer), J Knight (Editor), D Davies (Education Officer), C Acott, V Haller and R Walker (Committee Members).

Apologies

Drs D Gorman (Past President) and M Kluger (NZ Representative).

1 Minutes of the previous meeting (15/4/97)

Read and accepted as a true record after minor adjustments**.** Proposed J Knight, seconded G Williams.

2 Matters arising from the minutes

2.1 North American chapter update. Dr Williams will contact Jeffrey Bertsch with regard to his involvement in this.

2.2 SPUMS European representative update. Dr Henrik Straunstrup has kindly volunteered to be the representative.

2.3 Ex-Presidents Committee update. Dr Acott informed that the committee had discussed the "Return to Diving" presentation at the UHMS in Cancun. He did not feel that there was any need for the committee to meet in Adelaide in November.

2.4 Indemnity policy update from Dr Williams. This is still being researched.

2.5 Circularisation of the *Role of the convener* to Committee members. This will be done at the face to face meeting in Adelaide.

2.6 Update on purchasing of Oxygen equipment for the dive boats. One of the units is held in New Zealand by the New Zealand Chapter. The exact location of this unit will be discussed with Dr Mike Davis. Dr Williams will research the costs and benefits of purchasing a second unit. The Treasurer feels that purchase of a second unit should be put on hold at present. These units should be taken to the ASM in Palau in 1998. The advantage of taking other resuscitation equipment to Palau was discussed.

2.7 Update on the IBM version of the index of SPUMS journals. This can be purchased for $10 on disks and is in Word for Windows as a tab separated document.

2.8 Update on the inventory of SPUMS equipment and furnishings held by committee members. All committee members are to hand in a detailed

account of all old and new equipment to the Treasurer in Adelaide. This should include all computer software as well as hardware.

2.9 Update of SPUMS on the Internet. This will be discussed in detail in Adelaide.

2.10 Update of SPUMS forms.

2.10.1 Introductory information: needs to be revised.

2.10.2 Application for the Diving Doctors List (DDL): further approved courses need to be added to the list and it needs to show which courses are of 10 or more days duration.

2.10.3 SPUMS membership application form: this needs to clearly state the country of origin and have room for extra information such as facsimile and e-mail.

Committee members are to present their suggestions at the next meeting.

2.11 Funding of DES. DAN SEAP is funding the DES phone line. Donations are still being sought.

2.12 Duration of Committee positions. This will be fully discussed at the next meeting.

2.13 Project Proteus and the Ex-Presidents Committee position with regard to this. Dr Williams will discuss this directly with Dr D Walker. A letter of expression of interest in being involved in an asthma study will be circularised to Doctors on the Diving Doctor List.

3 Annual Scientific Meetings

3.1 1998 ASM Palau update. The conference booklet has been circulated to the Committee. All is going smoothly.

3.2 1999 ASM Layang Layang update from Dr Williams after the site inspection. Dr Kluger is no longer able to convene this meeting. Dr Acott has volunteered to convene the meeting, with Dr Williams assisting him. The site inspection was satisfactory although some of the rooms were noisy and the resort will need to address this. The meeting is planned for early May. As yet we are not sure of the dates of the UHMS Annual Scientific Meeting.

3.3 Suggested future ASM venues. For 2000 perhaps Fiji, Castaway Island. Further discussion at the next meeting.

3.4 Closure of the ASM NZ account. To be discussed with Dr M Davis.

4 Treasurer's report

A report on the financial status of the society was presented by Dr Wong.

5 Correspondence

5.1 DAN letter dated 29 July 1997. This was circulated for information.

5.2 DAN injury insurance details.

5.3 Townsville General Hospital (TGH) Hyperbaric Medicine Unit letter. The TGH course should be added to the DDL application form as an approved course. Details of course dates should be supplied to the Editor and if supplied will be printed in the Journal.

5.4 Republic of South Africa Institute of Maritime Medicine letter. This request for recognition has been passed to Dr Davies the Education Officer for assessment.

5.5 Standards Australia letter requesting name of SPUMS 1997 representative for the CS/83 Recreational Underwater Diving. The representative will be Dr Catherine Meehan.

5.6 Dr Knight letter re SPUMS representative on Standards Australia Committee CS/83 Recreational Underwater Diving. Dr Knight tenders his resignation from the above committee.

5.7 Dr R Moffitt letter re DDL**.** This has been attended to.

5.8 Dr M Kluger will be unable to convene the 1999 ASM. Dr Acott has volunteered to do so.

5.9 Dr Mike Davis letter. This is to be discussed at the next meeting.

5.10 Dr P Chapman-Smith letter. Dr Meehan to write to Dr Chapman-Smith to clarify the situation.

6 Other Business

6.1 SPUMS membership database will be discussed further at the next meeting.

6.2 Office 97 and update to Microsoft Access 97 for DDL . It was decided to update the SPUMS computers to Office 97. Steve Goble, who maintains the DDL, may also need an upgrade. Extra training will be sought by the Treasurer and Secretary in the programs they are using to facilitate better understanding and use of these programs. Drs Williams and Wong will further discuss the handling of the mailing list and transfer of this to the printers.

6.3 Requirements for the SPUMS list of members. To be further discussed.

6.4 Job descriptions for the Secretary, Treasurer, Education officer, Public Officer, Editor and Convenor to be presented at the next meeting.

6.5 The face to face committee meeting will be held in Adelaide on the weekend 1st-2nd November. The meeting will be held at the Hyperbaric Unit of the Royal Adelaide Hospital. It has been suggested that in future the face to face meeting should coincide with the HTNA meeting. This is to be discussed further.

Closed at 1145.

BOOK REVIEWS

BEATING THE BENDS. THE DIVERS GUIDE TO AVOIDING DECOMPRESSION SICKNESS

Alex Brylske

Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.

Price from the publishers $US 15.95. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370.

Key Words

Book review, decompression illness, tables, training.

The author of this book of 120 pages is the technical editor of *Dive Training* magazine and, according to the back cover of the book, is a world renowned instructor trainer. His reputation has been gained as the innovator of a number of educational programs for divers. If this book is reflection of the man's standard as a teacher, I would love to attend one of his courses.

The six chapters are set out in logical fashion and each has a rather catchy title. Chapter two for instance, which examines dive tables and their origins, is titled "If you want a guarantee, buy a washing machine" and chapter four is titled "Turning on a computer shouldn't mean turning off your brain".

This book is designed to be read by divers rather than diving physiologists or doctors, so that even I can understand the subject. It fulfils this goal admirably. Whereas most books on this subject are either so weighty that only researchers can understand them or so simplistic that the average diver gains absolutely nothing from them, this book manages to tread the middle path. It gives the reader a very good overview of the subject with plenty of detail but in an easily readable style. The author is to be commended.

The discussion on the history of the development of the dive tables, and on how to use the dive tables, is probably as good as I have read anywhere.

If I set out to find some deficiency in the book it would only be with the proof reading. Certainly the author has used his spell checker but it cannot differentiate between cord, as in piece of string, and chord, as in group of musical notes, when discussing the spinal cord. This minor irritation seemed to be limited to chapter one.

This book should probably be compulsory reading for all divers and should be used as a handbook by all instructors. I strongly recommend it to all SPUMS members.

David Davies

A PICTORIAL HISTORY OF DIVING

Edited by Arthur J Bachrach, Barbara M Desiderati and Mary M Matzen.

Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.

Price from the publishers $US 97.00. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370.

Key Words

Book review, history, equipment.

It has long been the practice that the introductory chapter in many books on diving list a simple history. This is true of even such formal publications as the *US Navy Diving Manual* as well as the excellent section in Eric Kindwall's book *Hyperbaric Medicine Practice*.

Diving history societies, which have formed a loose association, have been started around the world. These enthusiastic groups of diving historians have offered a large enough niche for specialist publications. They want complete, detailed information. *A Pictorial History of Diving* was one of the first books to cover the subject in detail. I have owned a copy for some time and since first reading the book I have found it an excellent reference. Best Publishing Company has placed it among its "Collector's Classics".

The book was conceived by the Undersea and Hyperbaric Medical Society (UHMS) to assist in the many requests for information and photographs on diving history from members and others. As would be expected from UHMS, the list of chapter authors reads as a Who's Who of diving. All are experts in their fields and have come together to produce this excellent book. Having multiple authors usually means that each chapter stands alone and chapters can be read in any order. That is certainly the case in *A Pictorial History of Diving*, but careful editing has ensured no overlap or duplication.

As the title implies it fulfils the promise of an illustrated history on the evolution of diving equipment from the ancient breath holding days through to the modern deep mixed gas saturation systems. The photographs and drawings are well supplemented by the accompanying text. The text fills in many revealing aspects about the development of diving equipment. Many of the authors were involved in these developments, so they have a perspective that has importantly been recorded.

It is not a new publication, having been printed in 1988, but it has stood the test of time. Most diving libraries have many books that are not current but that does not diminish their place in the collection. It may be time for a

reprint, but often when that happens changes are made and some aspects are omitted, replaced by more current content, which leads to a loss of information.

There are many great photographs but, as a historical text, more detailed captions are required for some photos. For example on page 15 fig 2-26, what is the helmet that the diver is wearing? As time goes by the answer will become harder to find. It could be a Dräger 12 bolt DM200, has anyone got any other ideas?

A Pictorial History of Diving remains one of the better books that review the technology and romance of diving in a comprehensive fashion. Indeed it is one of the very few texts that does address the general topic well.

Bob Ramsay

SCUBA DIVERS SIGN LANGUAGE MANUAL
James P Smith and LeAnn G Smith.
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from the publishers $US 12.95. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370.

Key Words
Book review, communication.

James Smith is a Certified Interpreter for the Deaf and a diver. LeAnn Smith is a sign instructor and interpreter. They have produced what will be a useful book for divers and their buddies who wish to communicate during a dive and are willing to learn 160 signs. The signs have been adapted from the signs used by the hearing impaired in the USA. According to the authors the signs are easy to learn, easy to use and easy to understand. Each sign is clearly illustrated and numbered.

The signs are for creating pictures with meanings. For instance the right hand can be a diver moving (swimming) into the open left hand (a cave). Of course learning the signs takes time and practice. The authors recommend using one's voice while making the signs as an aid to learning.

Chapter 1 covers signs important to survival underwater, those to be used in an emergency and those which may prevent an emergency. Chapter 2 is "look-like" signs while chapter 3 is "act-like" signs. Both of these chapters owe a lot to above water signing and some appear to be less than useful for a recreational diver, such as those for a number of tools and for closing and opening a door. Chapter 4 is position signs and chapter 5 is other useful signs such as "what is the time?" and the replies. Numbers are more complicated than holding up one to ten fingers, each number has a specific signs from zero to 9 which is much more useful than counting many fingers.

The final sign (number 160) is "I do not understand" which can be used as a question mark when combined with other signs to form questions such as "How long have we been down?" or "How much air have you got left?". Two vitally important questions which usually mean swimming over and grabbing wrist or contents gauge.

The index contains 233 words, arranged alphabetically, with the appropriate numbers for the signs to be used. *What, when, where, which* and *how* are all covered by sign 160. Unfortunately *how* has been given 159 as its sign in the index.

Two copies of this book will make a good Christmas present for your regular buddy and leave you one for yourself!

John Knight

DIVING MEDICAL CENTRE

SCUBA DIVING MEDICAL EXAMINER'S COURSES

A course for doctors on diving medicine, sufficient to meet the Queensland Government requirements for recreational scuba diver assessment (AS4005.1), will be held by the Diving Medical Centre at:

Bond University,
Gold Coast, Queensland.
10th-12th April 1998 (Easter Holidays)

Previous courses have been endorsed by the RACGP (QA&CE) for 3 Cat A CME Points per hour (total 69)

Phone Brisbane (07)-3376-1056 for further details

Information and application forms for courses can be obtained from

Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road
Jindalee, Queensland 4047
Telephone (07) 3376 1056
Fax (07) 3376 1056

SPUMS ANNUAL SCIENTIFIC MEETING 1997

PULMONARY BAROTRAUMA A NEW LOOK AT MECHANISMS

James Francis

Abstract

A review is presented of pulmonary barotrauma (PBT) of ascent in the context of the more general condition of pulmonary over-inflation and lung rupture. Having identified some caveats with respect to the diagnosis of the condition and discussed the routes which air may take having left the intra-pulmonary air spaces, the epidemiology of the condition in the contexts of diving and submarine escape is discussed, noting that gas embolism appears to be a frequent consequence of PBT. Predisposing factors such as inexperience, rapid ascents and pulmonary disease are discussed before comparing the presentation of PBT in diving with that arising in aviators, with mechanical ventilation of the lung and spontaneously. Of particular interest is the observation that gas embolism is a rare complication of PBT in circumstances where the victim is surrounded by air rather than water. Having briefly discussed some relevant pulmonary mechanics, possible mechanisms whereby lung rupture in divers appears to differ from other circumstances are presented. A common breathing pattern of divers (skip-breathing) and the increase in thoracic blood volume which occurs with immersion and negative-pressure breathing are considered to be possible explanations.

Key Words

Physiology, pulmonary barotrauma.

Introduction

It is frequently difficult to establish an accurate history and firm diagnosis in people who have suffered accidents underwater. In some, barotrauma has only become obvious many hours after the last dive when precise memories of critical details have faded. In others, events have been so dramatic and rapid that patients have been unable to recall exactly what happened during their ascent. Furthermore, a rapidly evolving neurological syndrome after diving has all too frequently been blamed on PBT and arterial gas embolism even where there has been no evidence of lung injury and where assessment of the dive profile indicates that the condition is just as likely to have been a consequence of a dissolved-gas disease process. As a result, there is an aura of mystery surrounding PBT and diving. In this presentation I will review a number of aspects of pulmonary barotrauma in order to identify why it is that the lung may rupture in certain circumstances. In doing so I would like to acknowledge the substantial contribution of Professor David Denison to this work. He and I have spent many a long hour debating these issues. This paper and the more substantial book chapter from which it is drawn[1] represent a synthesis of our experiences and thoughts.

Definitions

Pulmonary barotrauma (PBT) is a collective term for two different insults to the lung. The first may occur during exposure to increasing ambient pressure (PBT of descent, or "lung squeeze") and the second during exposure to reducing pressure (PBT of ascent). The former condition is extraordinarily rare to the point that PBT has come to be synonymous with PBT of ascent. Its rarity is of interest. During the descent phase of a breath-hold dive, the thoracic gas volume (TGV) will decrease in accordance with Boyle's Law. Assuming that the lung was filled to total lung capacity (TLC) immediately prior to the dive, the depth at which this will be reduced to residual volume (RV) can be expressed as 10*(TLC/RV -1) msw. For most people this would occur at a depth of between 30 and 50 m (of sea water). If this depth were exceeded, the compliance of the lung and thoracic wall would increase, resulting in the generation of a relatively negative pressure in the airways and barotrauma of descent would result.

However, the world record breath-hold dive of 127 m, currently (April 1997) held by Pipin Ferreras indicates that, unless he has a TLC/RV ratio of 13.7 (which is unlikely), the assumptions behind such a calculation are wrong. It has been suggested that the thoracic blood volume (TBV) increases during breath hold dives and that, by effectively reducing the residual volume, this effect would increase the theoretical maximum depth of such a dive.[2] Evidence to support this idea was provided by Schaefer et al.,[3] who carefully studied a series of breathhold dives by Robert Croft to a maximum depth of 73 m. With a TLC of 9.1l and a RV of 1.3 l, his theoretical maximum depth was 69.5 m. They found that, depending upon the depth of the dive, the TBV increased by between 850 and 1,047 ml. In the same report, they calculated that for Jacques Mayol (TLC 7.2l; RV 1.88l) to perform his (then) record dive to 70 m, an increased TBV of 980 ml would have been required. It seems that this effect explains the extraordinary depths to which breathhold dives can be conducted without pulmonary injury and that factors other than lung squeeze limit the maximum depth which can be achieved by divers with normal lungs.

I will focus on PBT of ascent. Vertical movement through a column of water is associated with substantial

changes in pressure. Since the liquid and solid tissues of the body are effectively incompressible, such pressure changes are evenly distributed between these tissues and there is little alteration in their function. Indeed, very high pressures can be tolerated before there is any resultant tissue dysfunction.[4]

The situation is different for tissues which contain, or are adjacent to, a gas phase because this is liable to the constraints of Boyle's Law. For some organ systems, such as the bowel, the contraction and expansion of its gaseous contents during increases and decreases in depth are normally of no consequence. This is because the bowel wall is highly compliant and, provided that no gas is introduced at depth, such changes in volume can normally be accommodated with an insignificant pressure differential being generated across its wall. However, where tissue compliance is limited, alterations in the volume of gas will be accompanied by the development of a more substantial pressure differential between the tissue and its adjacent gaseous phase. Furthermore, if gas is introduced into the space at depth, it will expand during ascent. Unless this gas-containing space is allowed to vent freely, a pressure differential will develop between it and the surrounding tissue. This force may result in tissue injury which is known as barotrauma of ascent.[5]

With respect to the lung, there is evidence that the degree of overpressure required to cause tissue injury is dependent on the extent to which the lung is splinted by its surrounding structures.[6-8] More strictly, therefore, the tissue injury of PBT is caused by over-stretching of the lung by a transmural pressure change rather than the change in volume of the intra-thoracic gas.

Distribution of extra-pulmonary gas

Once the lung has ruptured, gas is free to escape from the airways into surrounding tissues. Although the presence of aberrant air in the pleural, mediastinal or adjacent subcutaneous spaces, or in systemic arterial blood, is usually accepted as evidence of lung rupture, this is not necessarily so.

a Firstly, extensive ruptures of the lung interior from blast injury do not lead to the escape of much air unless the victim survives to make several to many respiratory efforts.[9-14]

b Secondly, gas of non-pulmonary origin can appear in peri-pulmonary tissues (e.g the pleural, pericardial, mediastinal, peritoneal and retro-peritoneal spaces, the soft tissues of the neck and chest wall and the pulmonary and systemic vasculature) because:
   i it has been generated *in situ* by infecting organisms,
   ii swallowed air has been forcibly expelled from the GI tract, as in the ruptured oesophagus syndrome described by Boerhaave[15] and Agarwal and Miller.[16]
   iii it has escaped from an extra-pulmonary airway in the mouth, pharynx, larynx, trachea or extra-pulmonary bronchi, and may have extended downwards to the mediastinum, the pericardium, burst through the thin hilar pleura to the pleural space or carried on down below the diaphragm.[17,18]

c Thirdly, some penetrating injuries of the chest wall and much cardiothoracic surgery lead to an open traumatic pneumothorax with considerable or complete collapse of an intact lung. Closed heart bypass surgery can lead to the accidental introduction of gas emboli via bypass lines or via oxygenators (bubble- more than membrane-oxygenators).[19-21]

d Of particular importance to the investigation of diving accidents, gas absorbed during the hyperbaric exposure which preceded the ascent to the surface may leave solution and form pockets of gas in almost any location. This can occur post-mortem (Calder IM, personal communication).

Lastly, as a diagnostic feature of PBT haemoptysis may be unreliable. Blood in the airways or in sputum often comes from broken vessels in the upper respiratory tract, especially the nose and the peri-nasal sinuses. It is helpful to keep these reservations in mind when reading reports of presumed lung rupture.

Common forms of lung rupture occur when pulmonary air:

a has ruptured into the pleural space via the visceral pleura, from whence it may extend into the chest wall, particularly if an inadequate drain is in place (spontaneous or closed traumatic pneumothorax).

b has been forced through the wall of a proximal airway, travelling centripetally along the bronchovascular bundles, to reach the mediastinum (spontaneous pneumomediastinum, mediastinal emphysema) from where it can travel up into the neck (cervical emphysema), out along superficial fascial planes (subcutaneous emphysema), down into the pericardium or sub-diaphragmatic spaces (pneumopericardium, pneumoretroperitoneum, pneumoperitoneum), or burst through the thin walls of the pulmonary arteries or veins, (pulmonary and/or systemic gas embolism).

c has escaped though the walls of, usually many, distal airways into the small bronchovascular bundles, splinting the lung, as it travels centrally (pulmonary interstitial emphysema or PIE). In this condition it is increasingly difficult to breathe, or to ventilate the lung artificially, as the air-filled splints extend. A striking account of this condition, developing during spontaneous breathing, is given by Torry and Grosh who cared for

and reviewed a vast number of U.S. soldiers affected by the virulent influenza epidemic of 1918.[22] They observed that many of these patients developed PIE, seen radiographically, that progressed to pneumomediastinum and cervical emphysema which often blocked venous return. Relief of the cervical obstruction was life-saving, (see also Coelho[23]).

Following lung rupture, the escaped gas may track in a number of directions as shown in Figure 1. In the case of hyperbaric decompression, it is difficult to assess the true frequency of the various manifestations of PBT for a number of reasons:

a Not all cases are symptomatic. Even where gas embolism of the brain has occurred, the manifestations may be minor, transient and go unreported.

b In all but the most trivial of hyperbaric exposures it is impossible, in most cases, to determine with confidence whether neurological manifestations have arisen as a result of arterial gas embolism from PBT or as a result of bubbles formed in tissues supersaturated with gas.

c In many parts of the world the collection of diving accident data is haphazard and therefore incomplete.

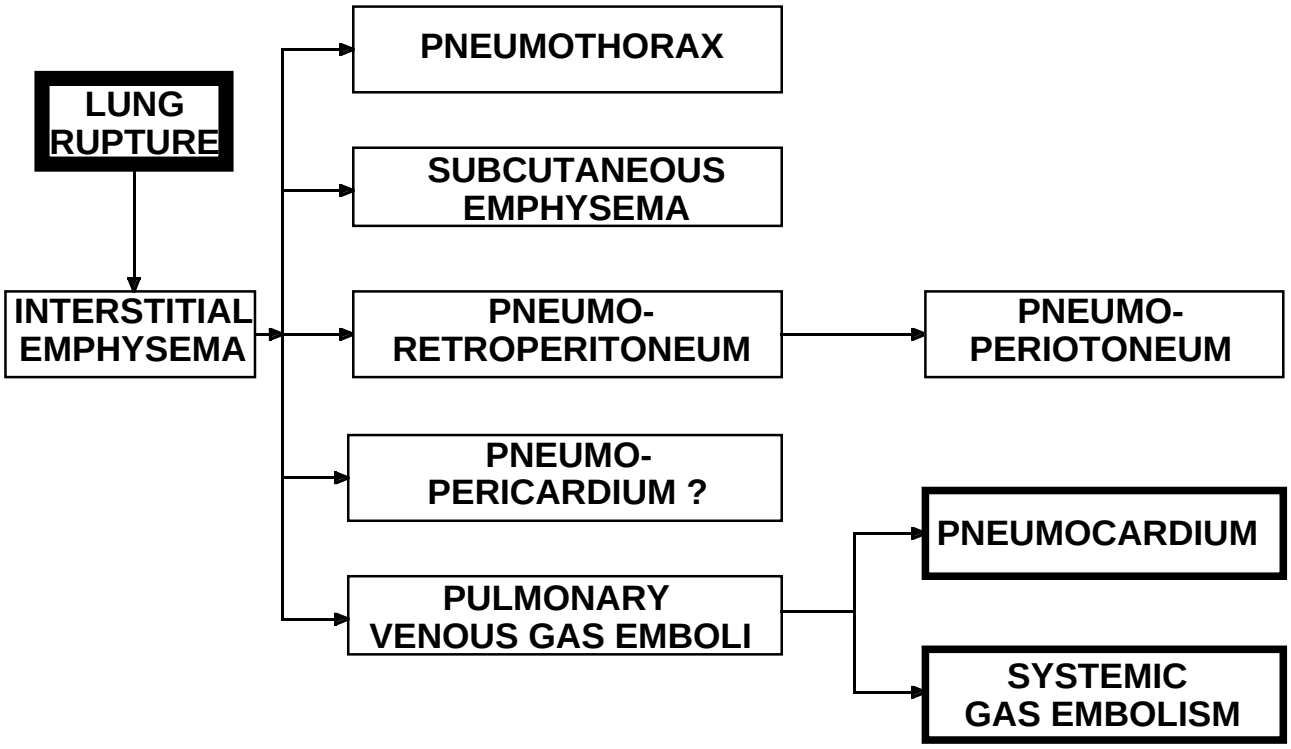


Figure 1. Possible routes for extra-pulmonary gas following pulmonary barotrauma.

TABLE 1

INCIDENCE OF PULMONARY BAROTRAUMA AND SYMPTOMS

Diagnosis	Benton[24]		Leitch[25]		Gorman[26]	
PBT only	8	9%	8	10%	42	66%
Neurological DCI	63	69%	60	77%	21	33%
Both	20	22%	10	13%	1	1%
Totals	**91**		**78**		**64**	

Under each investigator's name the first column gives the number of subjects and the next column gives the percentage of subjects with the diagnoses in the left hand column.

One specific circumstance where the latter two constraints do not apply is submarine escape training. Analysis of a series of 91 cases in which the diagnosis could be established with reasonable confidence[24] showed that pulmonary barotrauma alone occurred in eight, neurological DCI in 63 and pulmonary barotrauma with neurological DCI in 20 . PBT in this series was defined as cases in which there was x-ray or clinical evidence of a pneumothorax or extra-alveolar gas in the lung parenchyma, mediastinum or other site to which gas may track.

In a study of 140 cases of PBT in divers reported to the Institute of Naval Medicine over a period of 20 years, and which suffers from all of the above constraints, Leitch and Green described 78 cases which met similar diagnostic criteria.[25] . Gorman reported a similarly constrained study of 64 diving casualties in Australia.[26] The diagnoses of the

cases in these three studies is shown in Table 1. It can be seen that neurological manifestations, arising from the distribution of the escaped gas to the pulmonary venous circulation and subsequent cerebral arterial gas embolism, predominate. Within the non-neurological categories of PBT, pneumothorax is uncommon, present in only 5% of cases in submarine escape trainees.[27] In fatal cases, the finding of a pneumothorax at autopsy needs to be interpreted with caution as, in certain circumstances, it may be a consequence of post mortem events. In the remainder, following the escape of gas into the interstitial tissues of the lung, its migration to the mediastinum may be continued, where sufficient gas has escaped, cephalad to the subcutaneous tissues of the neck and head, or caudally in the retropleural and retroperitoneal planes to form collections beneath the diaphragm, around the great vessels of the abdomen and in the pelvis.[28] Occasionally the radiological features may suggest a pneumopericardium although close examination of the films commonly shows that such gas lies between the parietal pleura and pericardium.[5] In particularly severe cases, a pneumocardium may be seen.[29] It is assumed that gas found in the left side of the heart has travelled along the pulmonary veins and that gas in the right side of the heart has travelled against the normal flow in the pulmonary arterial tree.

Predisposing factors

A high proportion of cases of PBT occurs amongst inexperienced divers.[30-33] In a large study of accidents arising from submarine escape training, the incidence of PBT amongst initial trainees is almost double that of requalifiers.[24] In the diving setting, it is well recognised that rapid ascent is a predisposing factor for PBT.[34] This may occur as a result of panic following, for example, the loss or failure of a piece of equipment or running out of air. Should such panic result in involuntary closure of the glottis, PBT will be the consequence for all but the shallowest of ascents. Such crises are largely avoidable and panic is less likely to arise in experienced divers.[35] A potential problem during the very rapid ascents of submarine escape training is that only a brief interruption to a controlled exhalation caused by, for example, coughing, sneezing or hiccupping, may be sufficient to provoke PBT. This is particularly so during the last few metres of an ascent when the rate of increase of thoracic gas volume is at its greatest.

To avoid pulmonary over-inflation it is important to breathe out during a rapid ascent. This is not an intuitive action, particularly if the diver believes that he or she is out of air, and has to be learned. It is therefore not surprising that inexperienced divers are at greater risk of PBT than those who are more experienced. The reason why trainee submariners appear to be at greater risk than requalifiers is less clear. All Royal Navy submariners undertaking escape training are carefully instructed and supervised in the water, with particular attention being paid to adequate exhalation during ascent. There is no obvious reason why this should be less effective in initial trainees than requalifiers particularly since such training is infrequent, meaning that on each occasion the lessons need to be relearned.

It is also apparent from submarine escape training that PBT may occur in personnel who have been witnessed to exhale, apparently adequately, throughout their ascent.[25,36] Therefore, there are likely be other factors which may predispose an individual to PBT.

It has been argued for many years that conditions which cause airways obstruction predispose to PBT and such arguments can be persuasive.[37,38] Over-distension of the lung distal to the obstruction may occur as the diver ascends. In the case of reversible airways disease, support of this hypothesis has been provided by alarming anecdotal reports of asthmatics who have suffered from PBT.[39] Whilst there is little dispute that asthmatics who are symptomatic should not dive, both on the grounds of the risk of PBT and their reduced exercise tolerance, the situation with respect to asthmatics in remission is controversial.[40]

As Edmonds observes,[39] even if an asthmatic diver enters the water asymptomatic, the underwater environment provides a number of potential triggers for an acute asthmatic attack:

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

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

c Inhalation of hypertonic saline through a faulty regulator;

d Breathing against a resistance (increased gas density, faulty regulator, low air supply).

As a result of such arguments, asthmatics have been excluded from military and commercial diving for many years and discouraged from sports diving. However, such exclusion is unenforceable in most jurisdictions. The consequence of this approach has been that there is no substantial data set from which an objective assessment of the risk of PBT in asthmatics, compared with non-asthmatics, can be determined.[41,42] Recent attempts to gather such data have been criticised for poor study design[43-45] or are limited to small pilot studies.[46] Nonetheless, they indicate that the risk of asthma provoking PBT may have been overstated in the past and, until high quality data are available, this issue will remain controversial.[47]

The evidence for an association between chronic obstructive pulmonary disease (COPD) and PBT is also

scant. This is for much the same reasoning as above and also because COPD, unlike asthma, is unusual in the generally young, fit population from which submariners and military, commercial and the great majority of sports divers is drawn. There is radiological and autopsy evidence, albeit anecdotal, that bullous disease may predispose to PBT.[36,48-50] However, small bullae and apical blebs are common and may avoid detection by routine radiography. Furtermore there is anecdotal evidence that uneventful diving may be possible with encysted pockets of intra-thoracic, extra pulmonary gas.[51]

The autopsy evidence from cases of fatal PBT needs to be interpreted with caution since pulmonary over-inflation can cause blebs and careful histological examination is required to distinguish these from pre-existing disease.[49] Nonetheless, it can be argued intuitively that poorly- communicating, gas-filled spaces in the lung are liable to be vulnerable to barotrauma of ascent, particularly if the ascent is rapid. It is for this reason that Jenkins et al[52] have recommended that patients with conditions which may predispose to bullae or subpleural blebs, such as Marfan's and Ehlers-Danlos syndromes, should be considered unfit to dive.

The complete obstruction of a small airway is unlikely to predispose to PBT since the distal lung is likely to be atelectic and, even if it were not, the process of descent and ascent through a column of water is unlikely to result in a net increase in the volume of trapped gas in a similar manner to the bowel. However, incomplete obstruction could theoretically do so, in a similar manner to bullous disease, by limiting the rate at which gas distal to the obstruction can leave the lung during ascent. Despite the attractiveness of this hypothesis, there is a dearth of compelling evidence to support it. Liebow et al. reported a case, which has been frequently quoted, of fatal PBT in a submariner undergoing escape training.[48] At autopsy, an irregular calcified mass was found in the left main bronchus and a second in a superior segmental bronchus of the left lower lobe. The latter was associated with bronchiectasis, from which it can be implied that there was substantial chronic airway obstruction. Of interest, however, is the fact that the extent of the PBT in this case was not confined to the left lower lobe or even the left lung, since a pneumothorax and substantial interstitial emphysema were found on the right side. Although the finding of interstitial emphysema associated with the bronchiectasis may be evidence that localised airway obstruction is a predisposing factor for PBT, the question is left open as to whether the affected lung is more vulnerable than that served by normal airways.

Unsworth described a case of tension pneumothorax which arose following a session of hyperbaric oxygen therapy.[53] The patient was subsequently found to have a neoplastic mass in the right middle lobe. The pneumothorax was effectively treated and there were consequently no acute autopsy findings. The attribution of the pneumothorax to airway obstruction due to the neoplasm has, therefore, to be speculative. The same argument applies to the case reported by Calder in which localised airway obstruction was implied from the finding of clinical and histological evidence of acute bronchiolitis.[49]

To further confound the role of involuntary airways obstruction, it now appears that indices of obstruction do not correlate with the risk of sustaining PBT. Benton et al. studied the screening spirometry data of a population of healthy Royal Navy submarine escape trainees who had suffered unequivocal PBT.[24] They confirmed the observation of Brooks et al[54] that a lower than predicted forced vital capacity (FVC) was associated with PBT rather than any measured index of obstruction.

There can be little doubt that pleural scarring can predispose to PBT, particularly pneumothorax.[8,48,49] However, this is again a common condition and liable, if limited in extent, to be missed on a routine chest x-ray, which in any case may not be required for sports divers. Since it is likely that many thousands of uneventful dives have been conducted by personnel with pleural scarring, the magnitude of the risk of PBT associated with this is likely to be small.

Lung rupture in other circumstances

Before going on to discuss how the lung ruptures, it is worthwhile studying circumstances other than diving in which the lung may rupture. Rapid decompression to altitude is an obvious place to start. Although the early work of Robert Boyle,[55] Paul Bert[56] and others identified hypoxia and decompression illness as limitations to hypobaric decompression, it was not until the early 20th century that strategies to overcome these problems, other than the provision of oxygen at ambient pressure, were developed. Haldane realised that the maximum altitude to which men could go when breathing pure oxygen at ambient pressure would be about 40,000 ft.[57] He also calculated that delivering oxygen at an overpressure of about 15 mm Hg would enable them to fly a few thousand feet higher.

Towards the end of World War II, the pressurised aircraft cabin was developed. This obviated the need for prolonged positive-pressure breathing and allowed aircraft to climb much higher. However it introduced two new problems, those of rapid decompression and of the brief but profound positive-pressure breathing regimes that would be required to bring aircrew back safely, if they lost cabin pressures suddenly at altitudes well above 45,000 ft. Thus air forces throughout the world embarked on a series of theoretical, animal and human studies on rapid decompression and brief but severe positive pressure breathing.

In essence these studies have shown that the human lung can be decompressed very rapidly indeed without coming to harm, providing that the glottis is kept open during the decompression. Heath reported a series of 86,916 decompression runs to altitude that were without significant adverse effects.[58] These included 771 rapid decompressions. Hitchcock et al. exposed 150 subjects to a total of 550 "explosive" decompressions.[59] Holstrom reported the safe outcomes of a large series of decompressions from an initial altitude of 10,000 ft to a final altitude of 40,000 ft in 1.6-2.3 sec.[60] Bryan and Leach did likewise for a series from 8,000 ft to 40,000 ft (565 mm Hg to 141 mm Hg) in 2.5 sec.[61] Ernsting et al. safely completed 6 decompressions on each of 3 subjects, from 8,000 ft to 40,000 ft in 1.6 seconds.[62] Holness et al. successfully decompressed two subjects from 22,500 ft to 60,000 ft and from 45,000 ft to 80,000 ft.[63] The rate of decompression in these studies works out as roughly twice the rate achieved in the last 10 m of a submarine escape training run.

Meanwhile actual decompressions in flight were providing more evidence that the human lung could survive quite rapid decompressions. For example Achiary et al[64] reported satisfactory outcomes to 15 abrupt decompressions with time constants of 0.1 to 3 sec, in French high performance aircraft, and Brooks[65] did likewise with 29 incidents of massive/explosive decompression in Canadian fighter aircraft over the 20 year period 1962-1982. In almost every case the crew were able to land their aircraft safely.

Although millions of passengers, many of whom have respiratory disease, travel commercially each year, PBT is rare in flight or in decompression chambers. In Cran and Rumball's series of 994 spontaneous pneumothoraces occurring in RAF personnel over a 22 year period, only 8 occurred in flight.[66] This incidence was judged to be proportionate to the fraction of time spent in the air and was not thought to have been caused by the flights. Nonetheless, dramatic exceptions have been described in cases of pre-existing lung disease.[67,68] In contrast to decompression from depth, decompression to altitude rarely causes arterial gas embolism and only a single case report has been found.[69]

Another circumstance in which PBT can occur is with mechanical ventilation. There are several forms of mechanical ventilation. In one, gas is supplied to the respiratory tract at higher than ambient pressure, via an oronasal mask or an endotracheal tube (positive pressure ventilation). In a second, all of the body but for the head and neck is surrounded by a rigid vessel (or iron lung) which is cyclically decompressed whilst the airway is exposed to ambient air pressure, (negative pressure ventilation). In all but very minor details, these two forms of artificial ventilation have identical effects upon the lung and circulation. Both inflate the lungs but obstruct the circulation.[70]

In a third form, the chest wall alone is surrounded by a rigid jerkin which is cyclically decompressed whilst the rest of the body remains exposed to ambient pressure. This form of ventilation inflates the lung and promotes the circulation.[71]

Experiments on positive pressure inflation of fresh human cadavers show that lungs expanding in an unsupported thorax burst at about 70 mm Hg but, if the thorax is confined, they rupture in a different manner at pressures of about 110 mm Hg.[7,8] Many animal experiments have confirmed this.[72,73] In the early days of mechanical ventilation (with the Drinker negative-pressure iron lung) the technique was applied successfully for long periods of time at relatively low transthoracic pressures, about 30 cm of water, in patients with neurological disorders (usually polio) but healthy lungs. Since then, positive-pressure ventilation, often delivered by endotracheal tube, has been applied to very many people with damaged lungs (most frequently neonatal or adult respiratory distress syndromes). Not surprisingly, studies of such cases have shown that diseased lungs are more fragile than healthy ones. Diseased lungs often become stiff because of consolidation, loss of surfactant, fibrosis and/or pulmonary interstitial emphysema. Stiff lungs need higher pressures to ventilate them.[74] The pressure-volume curve of the diseased lung reflects the properties of the ventilateable part of the lung as has been demonstrated by the use of computed tomography.[75] Mechanical ventilation leads to pulmonary capillary rupture and to pulmonary oedema. These complications appear to be a function of lung volume rather than inflation pressure, so much so that Dreyfus et al. have proposed the term "barotrauma" should be replaced by "volutrauma".[76,77] Positive pressure ventilation opposes systemic venous return, causing pulmonary vessels to collapse, increasing the transmural stress on neighbouring alveoli.[78] Systemic hypovolaemia compounds this and may increase the risk of lung rupture.[79] Often a rise in pulmonary arterial pressure, i.e upstream of the compressed intra-pulmonary vessels, precedes a pneumomediastinum. Once the gas has escaped into the soft peri-pulmonary tissues (surgical emphysema) it can block cerebral venous return at the base of the neck.[23] It can also escape from the soft tissues into systemic veins leading to paradoxical systemic venous gas embolism.[80]

The commonest sequences observed in mechanically ventilated patients on Intensive Care Units are pulmonary interstitial emphysema (PIE) leading centripetally to pneumomediastinum and cervical emphysema and centrifugally to the appearance of sub-pleural pulmonary blebs. The appearance of blebs and/or surgical emphysema are ominous signs that a pneumothorax will soon follow. Sick patients tolerate pneumothoraces badly because they have little pulmonary reserve. In addition most pneumothoraces on mechanically ventilated patients proceed to tension pneumothoraces.[81,82]

In summary, many studies of lung damage during mechanical ventilation support the idea that the risk of rupture is rare at inflation pressures of 40 mm Hg or less and substantial at pressures of 70 mm Hg or more. Some show that respiratory gas can escape into the pulmonary arteries or capillaries and travel against the flow to fill the right heart with foam, or it can break into the pulmonary veins and travel with the flow to the left heart and to the coronary, carotid and cerebral circulations.

Finally, two common manifestations of PBT occur spontaneously. Spontaneous pneumothoraces have a trimodal distribution with patients' age, affecting neonates with respiratory distress, young adults with no other apparent lung disorder and patients over the age of 40 years who commonly have extensive, pre-existing lung disease. It is the middle group, of primary spontaneous pneumothoraces in young adults, who are of interest to diving medicine. Up to the early 1930's they were always attributed, without proof, to tuberculosis, but then Kjaergaard showed that they were rarely accompanied by any underlying lung disease.[83] Such primary spontaneous pneumothoraces may be caused by sudden, but not necessarily forceful, distortions of the chest wall, as in twisting to attach or find a seat-belt, but most develop when subjects are at rest. Sixty percent are noticed in the first three hours of the waking day.[84] They are much more frequent in young men than young women (in the ratio of 6 or 7 to 1) and they are very much more common in smokers than non-smokers. In young men "light" smoking increases the risk seven fold, moderate smoking raises it some twenty fold and heavy smoking about one hundred fold.[85,86] Almost all are unilateral and affect the right and left lungs with equal frequency, but some 2% are bilateral, probably betraying a defect in the mesothelial barrier separating the two pleural spaces in the antero-superior mediastinum. They rarely occur after the age of 40 years. Recurrence rates after recovery from a first spontaneous pneumothorax are high (about 50%), are more commonly ipsilateral than contralateral but are very infrequent after intervals of two years or more.[85,87,88]

Primary spontaneous pneumothoraces are believed to be due to the rupture of apical subpleural blebs that have probably filled with alveolar air dissecting from splits in local small airways. The apices are the sites of stress concentrations in the upright lung and it is thought this explains the higher incidence in tall, young men with tall, thin chests.[89] Such blebs are also found at the sharp edged apices of the lower lobes. The gravity-dependent density gradients seen in CT scans of healthy lobes suggest that, in the supine and prone postures at least, the major fissures act as shelves on which the uppermost lobe rests and from which the lowermost lobe hangs, so it is possible that there are important stress concentrations at these sites also.[90,91]

Some spontaneous pneumothoraces are associated with forced inspiratory manoeuvres such as hiccupping or the completion of tests of total lung capacity or peak inspiratory pressures.[92,93] Such cases suggest that voluntary high inflations can stretch some parts of healthy lungs beyond their elastic limits

Other clinical experiences at sea-level show that sudden and forceful expiratory efforts (e.g Valsalva manoeuvres as in child-birth, weight-lifting, playing the trumpet, violent coughing against an expiratory resistance or repeated attempts at producing a maximal expiratory pressure as a test of lung function can force air through the walls of the respiratory tract, most probably at extra-pulmonary or at extra-thoracic levels.[94-97]

Spontaneous pneumomediastinums are probably about 10 times rarer than spontaneous pneumothoraces. However, as many are asymptomatic and only half of them are visible on a straight postero-anterior radiograph of the chest, their true incidence is unknown. They are said to be present in 5% of all children X-rayed for asthma.[98,99] Often they are only suspected once dysphonia or cervical subcutaneous emphysema become obvious.[100] Their natural history and management are reviewed extensively by Pierson.[101] Recurrences are infrequent. The commonest presenting symptoms are: retrosternal pain (88%), dyspnoea (60%) and dysphagia (40%). Sixty percent also have cervical subcutaneous emphysema and one in eight have a concurrent pneumothorax.[102] In one retrospective study of 17 patients aged 12 years or more, 12 of the 17 gave a history of performing Valsalva manoeuvres during inhaled drug abuse.[103] Recently Fujiwara has reported spontaneous pneumomediastinum as a complication in 15% of patients with fibrotic lung disease.[104]

Summary of the circumstances for and consequences of lung rupture

The diving population, being predominantly male and aged between the late teens and early forties, is vulnerable to spontaneous lung rupture in the absence of any significant mechanical stress. When this happens it results in a primary pneumothorax or, more rarely, a pneumomediastinum. From a diving viewpoint, primary spontaneous pneumothoraces in the under 40 age group are unlikely to recur after an interval of two years. In the over 40 group, they are associated with pre-existing lung disease and are more likely to proceed to a tension pneumothorax and to recur. Since the diving population is generally fit, secondary spontaneous pneumothoraces in the over 40 age group is not a common problem. Traumatic pneumothoraces, in the absence of other lung disease, heal naturally and do not recur.

We know from experiments and experience with ventilators that normal lungs will rupture at an inflation pressure above about 70 mm Hg and that diseased lungs

will do so at a lower pressure. Again, the common presentations are of pneumomediastinum and pneumothorax while gas embolism is rare. Healthy lungs can be decompressed very rapidly to altitude over a pressure range of up to 0.5 bar without injury. If the lung is damaged, pneumothorax and pneumomediastinum are common and gas embolism is very rare.

In diving, the situation is different. Although very rapid ascents from great depth can be achieved without pulmonary injury, lung rupture can occur from a dive as shallow as 1 msw[105] in which the overpressure generated, assuming a breath-holding ascent at TLC, cannot be much greater than about 70 mm Hg. Although quantification is difficult, PBT in divers and submarine escape trainees appears to be more common and results in gas embolism far more frequently than in other circumstances, with pneumothorax being rare. In order to address why this may be it is necessary to review a little pulmonary anatomy and physiology.

Some relevant pulmonary anatomy and physiology

The pulmonary arterial tree lies directly alongside the bronchial tree, sharing a common capsule. The pulmonary venous tree lies between, but separated from, the arterio-bronchial bundles. As the trees approach the hilum the veins and airways come closer together. Most alveoli abut on each other but some lie directly alongside the broncho-arterial bundles that penetrate the lung. These marginal alveoli may be critical in some forms of lung rupture.[78] The healthy pleural cavity used to be thought of as a potential, rather than actual, space, but recent electron microscopic studies have shown it to be a continuous liquid film about 1 μm thick with a volume of a few millilitres.[106]

In water, the variations in ambient pressure between the uppermost and lowermost parts of a submerged water-filled sac, such as a lung-less human body, are considerable but balanced by almost equal hydrostatic gradients within.[107] In a gas-filled sac (Figure 2), such as the thorax, submerged to the same depth, the gas pressure within is determined by the net force pressing on the asymmetrically shaped vessel from without. It is that pressure which would exist at the sac's centroid of pressure (C_p), were it to be filled with water continuous with the water outside. Many experiments have shown that, for a typical adult male thorax, this point lies in the mid-sagittal plane some 19 cm below and some 4 cm behind the sternal notch, i.e. somewhere in the middle of the right atrium (Figure 3).[108] Gas delivered to the lung at this pressure, when the chest wall is relaxed, will inflate it to its normal functional residual capacity. This eupnoeic pressure is that at which the work of breathing underwater is minimised. By holding a regulator in his mouth, a diver who is vertical in the water is breathes at a relatively negative pressure.

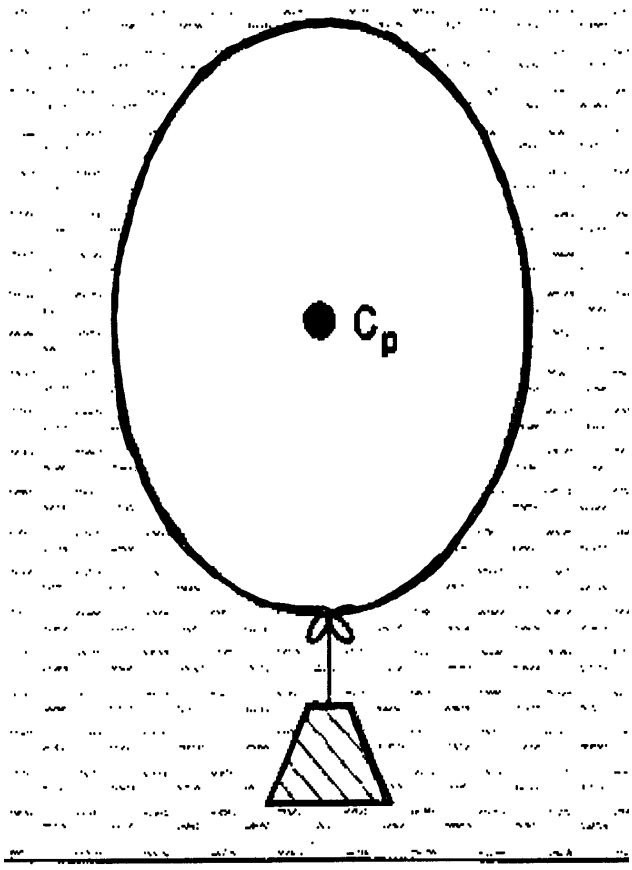


Figure 2. The centroid of pressure (C_p) of a weighted balloon immersed in water.

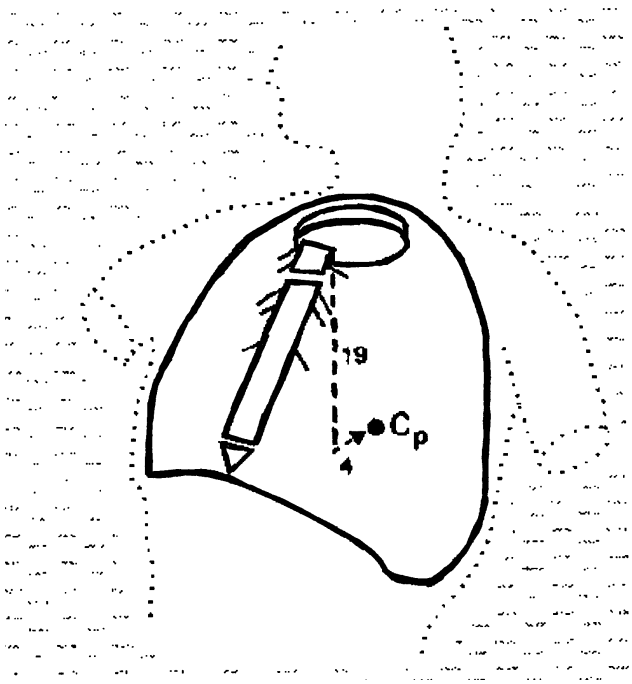


Figure 3. The centroid of pressure (C_p) of the human chest is located approximately 19 cm below and 4 cm behind the sternal notch.

Figure 4 shows an elastic, gas-filled balloon, modelling the lung, that empties through a floppy tube, modelling the airways, when they are compressed by the syringe or thorax. As the plunger is advanced the pressure immediately outside the balloon and tube rises. The pressure within the balloon is augmented by the elastic recoil pressure of the balloon at its volume at any instant. The pressure along the tube falls from the sum of $P_{syringe}$ and P_{recoil} at one end to $P_{ambient}$ at the other. It follows that at some point along the tube the internal pressure will equal $P_{syringe}$. Proximal to that **equal pressure point** the floppy tube will be inflated, distal to that point it will be collapsed (unless the airway at that point is rigid). The higher the value of $P_{balloon}$, the more completely the balloon will empty. At RV in adults, each airway will be closed at some point along its length. If this system is decompressed, the equal pressure point will move to the right as $P_{ambient}$ is reduced and the balloon will empty.

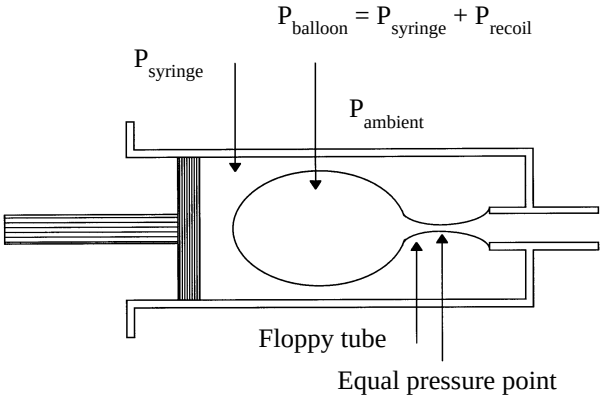


Figure 4. A balloon-in-syringe model of the lung showing airway collapse at the equal pressure point (see text).

The chest wall has a tendency to spring outwards and the lung has a tendency to collapse inwards. The elastin fibres of the broncho-alveolar tree are the prime determinants of the stretchability or compliance of the lung, i.e the instantaneous slope of its volume/pressure curve. This curve is sigmoid and eventually almost flat, indicating that its maximum compliance is in its mid-volume range. It becomes more rigid as it approaches residual volume and stiffens markedly as it approaches its maximum volume. The collagen fibres of the tree and pleura set the upper limit to its expansion.[109-111]

The inspiratory power of the chest wall is maximum at minimum lung volume and least at maximum lung volume. The reverse is true for its expiratory power. The maximum gas volume of the lung that can be achieved by a voluntary inspiration is described as Total Lung Capacity (TLC). It is reached when the diminishing inspiratory power of the expanding chest wall is balanced by the increasing elastic recoil pressure of the enlarging lung. Note that the lung itself can expand by a further 15% or so before it reaches its elastic limit. The minimum gas volume of the lung that can be achieved by voluntary expiration is described as its residual volume (RV). In children and adolescents it is set by the power of the expiratory muscles (mainly the abdominal wall) but if their chest walls are squeezed when they are at RV, more air can be heard to leave. In adults, RV is set by the collapsibility of the bronchial tree as bronchial closure proceeds reaching the elastic limit of the chest wall. When their chests are squeezed at RV no more air comes out although some 1.5 litres are still within.

In essence the lung hangs from the uppermost parietal pleural surface and sometimes rests on the lowermost parietal pleural surface, that is to say that the weight of the whole lung is concentrated at the apex of its upper surface (in any posture). The stress concentration that results is believed to favour the development of sub-pleural blebs of air that have escaped from alveoli which have fractured locally.[89,112]

On inspiration the expanding chest wall increases the negative pressure in the pleural film expanding the intra- and extra-pulmonary airways in the thorax, but the negative pressure conveyed to the gas in the airways is then transmitted to the extra-thoracic airways, which tend to collapse. On expiration, the compressing chest wall raises the pleural pressure causing the extra-pulmonary, intra-thoracic airways to collapse immediately (pressure-dependent collapse) and causing the intra-pulmonary airways to collapse progressively as emptying proceeds (volume-dependent collapse) until, at RV, all routes to the mouth are closed somewhere along their length.

In a forced expiration, peak flows rise to twice the FVC/second. After one second, about 75% of the vital capacity has been exhaled. This volume known as the forced expired volume in 1 second, or FEV_1, is frequently compared with the FVC, and this FEV_1/FVC ratio is used as an index of airway obstruction but, in relation to pulmonary barotrauma, this is misleading. When the positively pressurised lung empties during sustained decompression it does so from a near-maximal volume to which the peak expiratory flow (PEF) is more relevant. The PEF indicates that the normal lung at TLC will vent a volume equal to one vital capacity in about one-fifth of a second, i.e. the fully inflated respiratory system behaves as if it had an unimpeded time-constant of emptying of no more than 0.3 sec. The experimental work of Haber and Clamann suggests that this figure will be almost independent of depth.[113] The irrelevance of the FEV_1/FVC ratio in predicting those vulnerable to PBT in diving or submarine escape has been shown at the Institute of Naval Medicine over the past 15 years. Following a series of accidents in the Submarine Escape Training Tank in the early 1970s, the routine spirometric testing of submariners and divers was introduced. At entry into their relevant service they had to achieve an FEV_1/FVC ratio of 75% and subsequently 70%. This resulted in approximately 12% of new entrants being

rejected on spirometric grounds, an unacceptable wastage rate with the manpower reductions in the Navy of the early 1980s. To address this issue it was agreed in 1983 that, provided other aspects of lung function (notably transfer factor and lung volumes) were within normal limits and there was no evidence of gas trapping, candidates who failed their Vitalograph would be permitted to undertake submarine escape training or diving. The rate of rejection of candidates fell from 12% to less than 2%, with no increase in PBT rates for either group.

Proposed mechanisms for lung rupture in divers

We know that divers who fail to exhale during ascent, or do so inadequately, rupture their lungs. Although very occasionally cases are reported in the literature where a pulmonary lesion may have been the cause of the problem, there remains a group of divers and submarine escape trainees for whom, despite extensive investigation, no explanation for their PBT can be found. This highlights, amongst other things, the limitations of even the most sophisticated investigative tools which are frequently disadvantaged by being employed some time after the accident. However, since only a very small lung defect is required for it to rupture, it is considered unlikely that even investigation shortly after the incident would prove more fruitful. The following ideas are presented as possible explanations for these cases.

1 It is important to appreciate that the lung ruptures when it is stretched beyond its elastic limit. In some people, this can occur when a deep breath is taken and held. In normal life at an ambient pressure of about 1 bar (ATA), when the lung ruptures in these circumstances, it would appear that the gas escapes into the mediastinum. The tears in these instances must have occurred in the lung interior, otherwise they would have led to pneumothoraces and they must have been close to broncho-vascular bundles, otherwise gas would not have been able to reach the mediastinum. In diving, it is considered that this is more likely to occur and that the condition will be symptomatic. It is more likely to occur because divers commonly skip breathe in which the normal sequence of: inhale - exhale - pause, is changed to: inhale - pause - exhale. It can be seen that this technique exposes the lung to a greater period of time during which it is at, or close to, its elastic limit, thereby increasing the opportunity for it to rupture. Gas escaping from the lung at depth will expand in accordance with Boyle's Law during ascent to the surface. By effectively increasing the volume of gas which has escaped from the lung, this will increase the probability that symptoms will arise.

2 Why, as Colebatch and Brooks and Pethybridge proposed,[54,114,115] is rupture of the lung during diving more likely to occur in small, stiff lungs than in large obstructed ones? The Macklins' elegant ideas on the particular vulnerability of marginal alveoli, close to bronchovascular bundles, and the possible compounding effect of hypovolaemia should be noted.[78] This, they argued, could increase the stress on the marginal alveoli if the cardiac output could not increase, thereby allowing the bronchovascular bundles to expand at a similar rate to the gas-containing elements of the lung. However, David Denison and I wish to add two additional proposals:

a Figure 5 shows an imaginary spherical lung in which bronchovascular bundles and their associated collagen and elastin fibres radiate outwards from the centre of the sphere. As the normal lung is inflated, it expands isotropically and stress is evenly distributed along each bundle with no one more liable to rupture than the others. Now introduce fibrosis into the middle third of one element and inflate the lung again. The fibrosed part is stronger than the rest of the lung and therefore less liable to tear. But, because it also can not stretch, it puts additional stress on the normal elements in series with it, making them more liable than the rest of the bundles to rupture. This may explain Calder's observation that he could find no consistent association between the sites of lung rupture and pulmonary scarring.[49]

b A feature of the submarine escape trainees and divers, which differs from all the other circumstances in which PBT may occur discussed above, is that they are immersed in water. In the case of submarine escape trainees using either a submarine escape and immersion suit (SEIS) or a Steinke hood, they are invariably head-up and vertical in the water column and therefore subjected to negative-pressure breathing because their

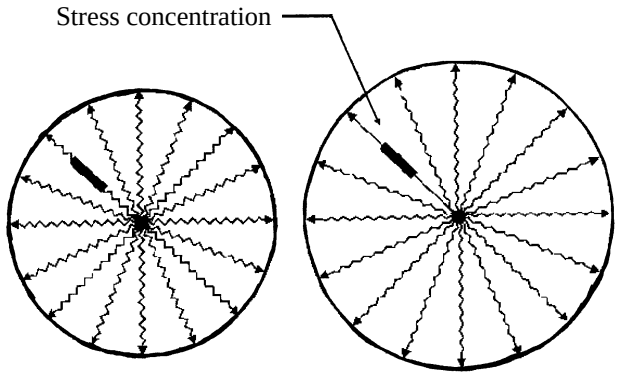


Figure 5. A model of the lung showing, on the left, a representation of the radial arrangement of the collagen and elastin fibres at functional residual capacity (FRC). The middle third of one of these is fibrosed and incapable of stretching. At total lung capacity (TLC), shown on the right, the stress is concentrated at the fibers which are in series with the fibrosis and it is here that they may rupture.

mouth is above the centrum of the lungs. For most divers (saturation divers being a notable exception), the same applies during surfacing and, in addition to the negative pressure breathing, additional negative pressure is commonly required each breathing cycle to trigger the regulator. Although the extent of the increase in TBV (discussed under lung squeeze in breath-hold divers) is less when breathing apparatus is worn underwater, there is still a displacement of blood into the thorax which occurs with submersion, particularly during negative pressure breathing.[116,117] This causes a reduction in compliance of the lung which is liable to make it more susceptible to tearing.

We have seen that, as the lung reaches TLC, its compliance is reduced and, in a setting of potential pulmonary over-inflation, it becomes more likely to rupture. It may be, therefore, that lungs of normal compliance, but which are smaller than predicted for an individual, are at a greater risk of tearing than normally-sized lungs because they are more readily overfilled. This may be an explanation for the findings of Brooks and Pethybridge et al. that a low FVC is associated with PBT rather than any spirometric index of obstruction.[24,54]

In most circumstances where the lung is exposed to an over-pressure, such as in mechanical ventilation, the over-pressure causes the pulmonary vasculature to empty. This may serve as a protective mechanism to prevent escaping gas from entering the pulmonary circulation. Immersion in water and negative-pressure breathing oppose this process, such that the TBV of divers and submarine escape trainees is liable to be greater at the point of lung rupture than in most other circumstances and most notably the military pilot subjected to positive pressure breathing during explosive decompression. Could this be an explanation for the far higher prevalence of apparent gas embolism arising from in-water PBT than from other causes?

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DIVING AND THE LUNG

Richard Moon and Bryant Stolp

Key Words
Physiology, pulmonary barotrauma.

Introduction

The respiratory system is affected by diving via a number of mechanisms. The increased flow resistance

engendered by breathing dense gas (Fig 1) and the additional mechanical load of the breathing apparatus added to the changes in pulmonary compliance caused by water immersion may significantly reduce ventilatory capacity. Dense gas breathing also engenders a greater likelihood of impairment of gas exchange due to diffusion problems in the alveolus. In addition, the lung is potentially subjected to damage during decompression by both pulmonary overexpansion and the effects of venous gas embolism. Finally, there is an uncommon syndrome in which young healthy individuals develop pulmonary oedema shortly after immersion at the beginning of a dive.

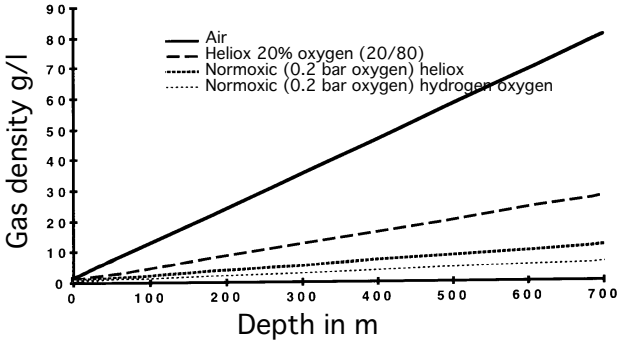

Figure 1. Gas density as a function of depth.

The densities of normoxic helium-oxygen ($He-O_2$) and hydrogen-oxygen (H_2-O_2) are displayed assuming a constant partial pressure of O_2 of 0.2 ATA. Gas temperature is 37°C.

external pressure gradient of the water column. This results in a redistribution of blood from the extremities into the thorax ranging from 500 to 800 ml. Some of this blood is retained within the great vessels and the heart, while a proportion of it engorges the pulmonary vessels, causing an increase in central venous and pulmonary artery pressures.[7] This results in a reduction in lung volume, particularly functional residual capacity (FRC)[8] and expiratory reserve volume (ERV), and a reduction in MVV of 5-10%.[1] When experiments are carried out in a hyperbaric chamber, immersion while the chamber is at pressure results in less decrement than at the surface.[9] The effects of immersion

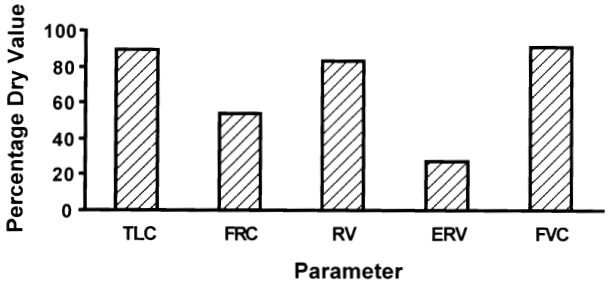

Figure 2. Effect of immersion on lung volumes.

Total lung capacity (TLC), functional residual capacity (FRC), residual volume (RV), expiratory reserve volume (ERV), forced vital capacity (FVC). Immersion reduces lung volumes predominantly by causing redistribution of blood from the legs into the thorax (see text).

Immersion

PHYSIOLOGICAL EFFECTS

During head-up immersion, the normal tendency for blood to pool in the legs due to gravity is immediately reversed as the hydrostatic pressure gradient within the venous system is almost exactly counterbalanced by the are fully exemplified by immersion to the neck and there is no additional load engendered by further descent into the water column. Changes in lung volume which occur on immersion are depicted in Fig. 2.

The engorged pulmonary vessels impinge upon the airways and increase airway resistance. Morrison and Taylor reported that subjects at rest experienced a 3 fold increase

TABLE 1
DENSITY OF BREATHING FLUIDS

Fluid	Depth (msw)	Density (g/l)
Air	0	1.1
$He-O_2$ (20% O_2)	0	0.4
Air	40	5.6
Air	50	6.8
Air	90	11.4
$He-O_2$ (20% O_2)	50	2.3
Trimix-10 (10% N_2 0.5 ATA O_2, balance He): (the highest gas density at which arterial blood gases have been measured during exercise)	650	17.1[1]
Ne-He-O_2 (0.21 ATA O_2, balance 76.8% Ne, 23.2% He): (the highest gas density breathed by man)	377	25.2[2]
Water		1000.0 [3-6]

in flow resistive work of breathing when they were immersed in water.[10] Upon immersion to the neck in water, pulmonary dynamic compliance (Cdyn) is reduced approximately 50% but static compliance (Cst) is unchanged.[11] The effect appears to be due to the immersion-induced reduction in lung volume that occurs because pressure at the mouth is lower than at the lung centroid. When mouthpiece pressure is increased to a value equal to the hydrostatic pressure at the lung centroid, thus restoring lung volume to control value, both dynamic and static compliance are returned to normal. Static compliance is a measure of the change in static lung volume for a given change in transpulmonary pressure ($\Delta V/\Delta P$) whereas dynamic compliance, measured during breathing or panting includes both respiratory compliance and airway resistance. The reduction in Cdyn with immersion is therefore probably due to the change in airway resistance and not due to altered lung tissue compliance secondary to engorged pulmonary vasculature.

Immersion also causes a tendency for airways to close at a higher lung volume (increased closing volume).[12-15] Airway closure during immersion tends to occur at lung volumes greater than FRC in older individuals.[15,16] It has been suggested that if closing volume is greater than functional residual capacity, gas exchange units subtended by closed airways would increase venous admixture, causing a reduction in arterial PO_2. Cohen et al. reported that alveolar-arterial gradient (P_AO_2-P_aO_2) increased from 7 to 16 mm Hg when subjects (mean age 23 years) were immersed to the neck in water.[17] However, a study in which blood gases and V_A/Q of the lung were measured, immersion caused neither an increase in shunt nor blood flow to low V_A/Q units nor a reduction in PaO_2.[14]

IMMERSION PULMONARY OEDEMA.

Immersion pulmonary oedema is a syndrome in which divers develop dyspnoea and cough productive of pink, frothy sputum shortly after beginning a dive.[18-20] Initially it was believed to occur only in cold water, which supported the observation that the normal increase in forearm vascular resistance upon cold exposure was exaggerated in affected individuals, several of whom subsequently developed hypertension.[18] However, the syndrome can also develop in warm water, and cold exposure does not always cause an exaggerated increase in forearm vascular resistance.

Although the cause is not fully understood there are several possible factors which could promote pulmonary oedema. The increase in pulmonary vascular pressures secondary to blood redistribution from the periphery to the central compartment is enhanced by exercise,[7] and probably also by cold induced peripheral vasoconstriction. It has been suggested that this increases airway resistance, which then augments the effects of dense gas breathing (see below) and the effect of external breathing resistance. During inspiration, when the intrathoracic pressure is more negative than usual, the left ventricular transmural pressure required to eject blood (afterload) is increased. A higher afterload on the left ventricle, when the pulmonary vasculature is already engorged due to immersion, could perhaps precipitate a critical increase in pulmonary venous, and hence capillary, pressure. This mechanism has been implicated in negative pressure pulmonary oedema during emergence from general anaesthesia[21] Finally, high vascular pressure in conjunction with elevated pulmonary blood flow has been hypothesised to cause direct endothelial damage and capillary leak due to high shear stress.[22,23]

The effects of increased gas density.

Density and viscosity are primary determinants of the resistance to gas flow through a pipe. While gas viscosity is not significantly altered by pressures within the range of human diving, there is a linear increase in gas density with ambient pressure. The theory of constant flow in an infinitely long tube predicts that resistance increases in direct proportion to density. Measurements in divers indicate that airway resistance is greater during expiration than inspiration, and increases approximately in proportion to the square root of the density.[24,25]

Under normobaric conditions exercise is typically limited by the functional capacity of the cardiovascular system. However, at higher barometric pressure, and hence gas density, exercise may be limited by the ability to move gas into and out of the lungs. One way of quantifying the effect of increased gas density on pulmonary capacity is to measure the maximum voluntary ventilation (MVV). This represents the total amount of gas per minute that can be voluntarily moved in and out of the lungs with maximal effort. MVV has been measured systematically over a range of depths and gas densities and its relationship to ambient pressure (in atmospheres absolute) can be described as follows (Fig. 3):

$$MVV_{ATA} = MVV_0\rho^{-k}$$

where: MVV_{ATA} = maximum voluntary ventilation at depth (measured as pressure in atmospheres abolute)

MVV_0 = MVV at the surface

ρ = gas density (g/l)

k = constant (0.3-0.5)

The mechanical effects of dense gas have been vividly illustrated by Drs Larry Wood and Charles Bryan, who performed isovolume pressure-flow measurements on themselves breathing air at the surface and at equivalent depths of 30 and 90 meters in a chamber (Fig. 4). At depth expiratory flow limitation occurs at lower transmural pressures and higher volumes when compared with surface controls. Since maximum expiratory flow is determined

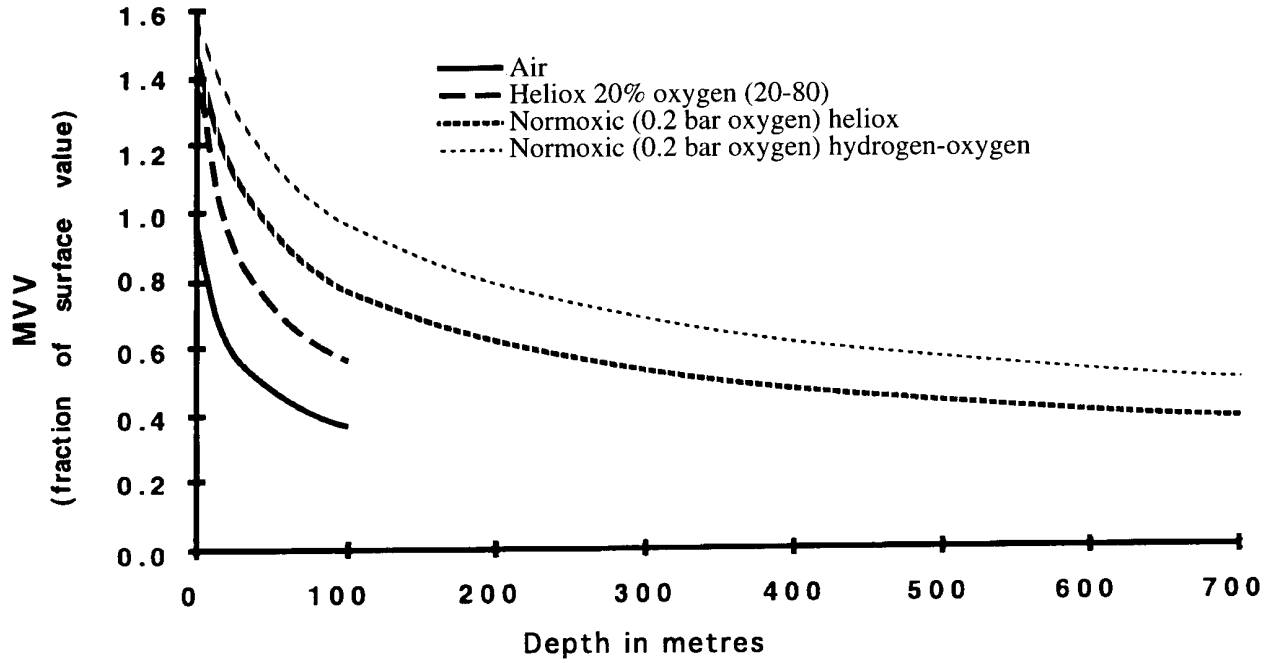

Figure 3. Effect of dive depth and breathing gas on maximum voluntary ventilation.

Air and heliox 80-20 are not shown deeper than 100 m as use of these breathing gases at deeper depths is limited by oxygen toxicity.

primarily by the elastic recoil pressure of the lung, this experiment illustrates the breathing strategy necessary to maximise ventilation at depth: increase lung volume and shorten inspiratory time to allow maximum time for exhalation. Expiratory flow-volume curves were recorded by the same investigators (Fig. 5).[26]

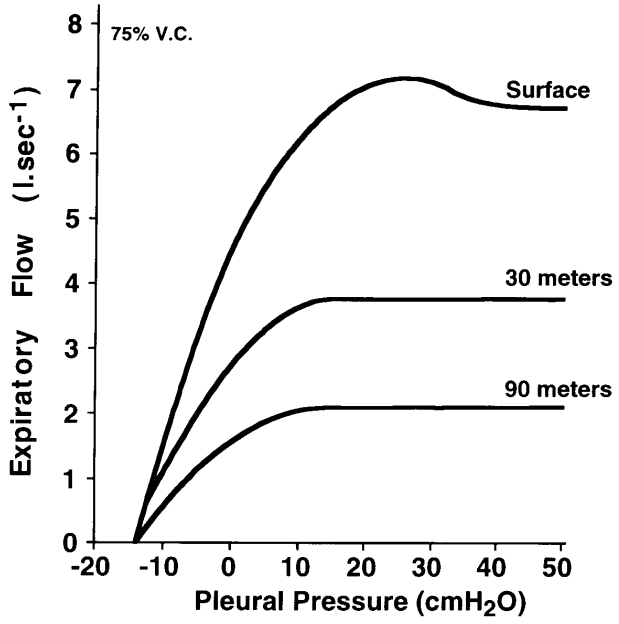

Figure 4. Isovolume pressure-flow relationship in one subject breathing air in a dry hyperbaric chamber as a function of depth (at 75% vital capacity).

There is a progressive fall in maximum expiratory flow, which remains constant at pleural pressures greater than 10-20 cm H_2O. Flow in this region is limited by dynamic airway compression, and can be explained by wave speed limitation.[69] Data shown are from Wood and Bryan.[70]

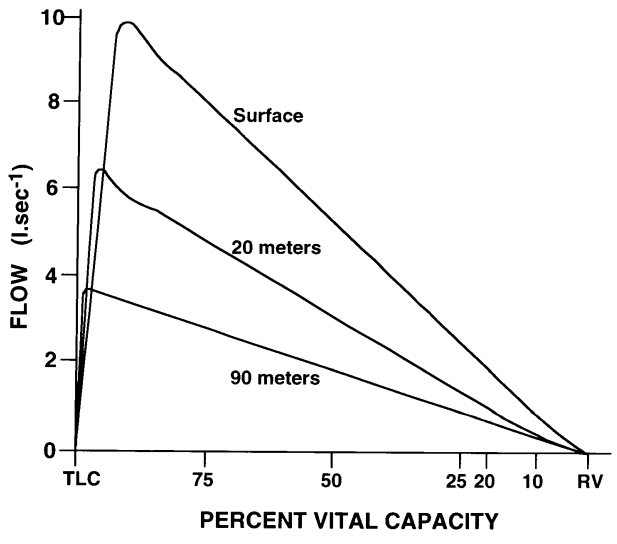

Figure 5. Expiratory flow-volume curves at various depths in a dry chamber breathing air.

Peak flow and the slope of the linear portion of the curve are highly dependent upon gas density. The data indicate that at least some density dependent (turbulent) flow exists in the lung under almost all conditions. Data from Wood and Bryan.[26]

Maximum expiratory flow rate and lung conductance (G, the reciprocal of resistance), have been measured over a range of gas densities (Figs. 6 and 7).

$G \propto \rho^{-c}$

where:

c is a constant (0.39 during tidal breathing and 0.47 during hyperventilation).

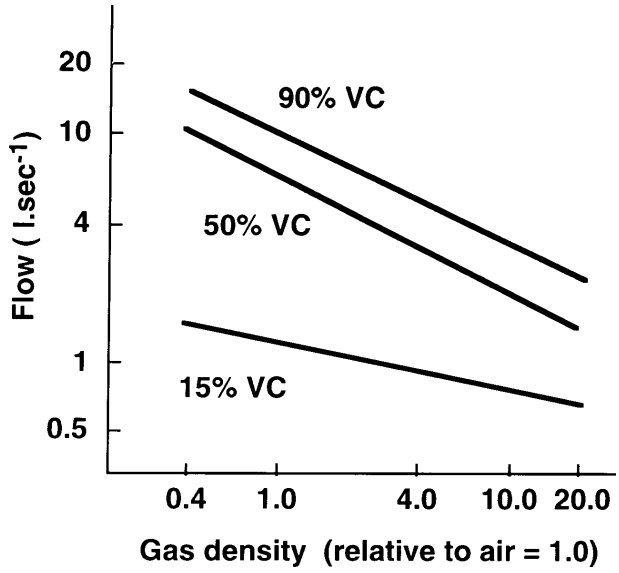

Figure 6. Maximum expiratory flow as a function of gas density and lung volume.

Data from Anthonisen.[24]

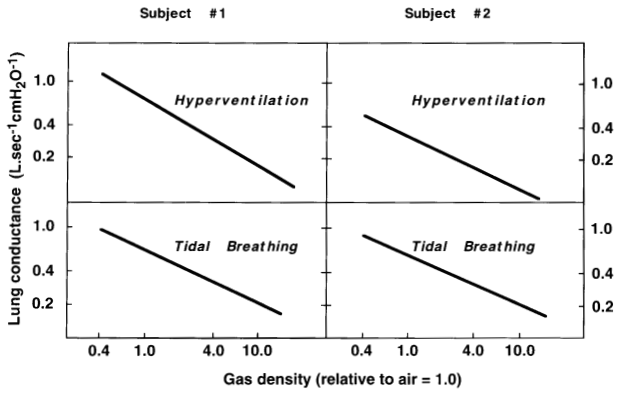

Figure 7. Lung conductance as a function of density in two individuals.

The lower panels represent inspiratory conductance during hyperventilation. During tidal breathing conductance (G) was proportional to $r^{-0.39}$; during hyperventilation G was proportional to $\rho^{-0.47}$, where ρ = gas density. The authors hypothesized that during hyperventilation flow was more turbulent and hence dependent on gas density to a greater degree. Data from Anthonisen.[24]

Resistance during inspiration is typically lower than it is during expiration (Fig. 8), suggesting that in order to achieve maximum ventilation (or minimise resistive work of breathing) a diver should use a short inspiratory time and breathe at a high lung volume.

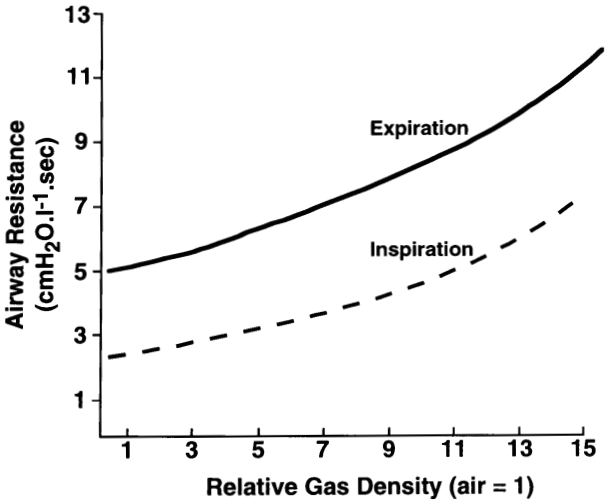

Figure 8. Inspiratory and expiratory flow resistance as a function of gas density during voluntary hyperventilation.

Because of increased airway calibre during inspiration, inspiratory resistance is less than expiratory resistance. These data indicate that maximum ventilation is highly dependent upon the ventilatory strategy chosen by the diver. A short inspiration and prolonged expiration, carried out at high lung volumes will minimise the mechanical load imposed by high breathing resistance. Data from Vorosmarti.[25]

At the surface the maximum exercise ventilation is typically about half of the MVV. At 20 m depth (3 bar or ATA) breathing air, MVV is reduced by about 35 percent; at 40 m the MVV is reduced to about 50 percent of its surface value. Thus maximum exertion at depths in excess of 40 m is likely to be associated with relative hypoventilation as the ventilation required to eliminate metabolically produced CO_2 exceeds the maximum possible ventilation. This analysis tends to underestimate the predicted maximum exercise rate because maximum ventilation during exercise is approximately 10% higher than at rest,[27] and the respiratory control mechanism in exercising divers allows their arterial PCO_2 to rise.[1,28-30] On the other hand these factors may be offset by the increase in physiological dead space (see below).

However, the MVV may be an inaccurate predictor of maximum exercise capacity. The short term MVV does not require sustained respiratory muscle effort, as does the increase in ventilation required for exercise, and the

maximum sustainable ventilation is only about 50% of the 15 second MVV.[31,32] Stolp[33] and Shephard[34] attempted to predict maximum exercise ventilation as a function of sustained ventilatory capacity (SVC: sustained isocapnoeic MVV >3 minutes in duration) at high gas densities and found that when exercise ventilation exceeded 45-60% of SVC there appeared to be a respiratory limitation to exercise.

During diving exercise ventilation tends to be lower than at the surface, which can contribute to hypercapnoea.[29,35] While it would appear self evident that this is due to high airway resistance, some evidence suggests that it is ambient pressure rather than density that predicts hypercapnoea. Salzano, during simulated chamber dives at depths up to 650 m, actually observed higher ventilation during moderate exercise (see Fig. 9).[1]

While airway resistance may play a major role in determining ventilatory performance during diving, one must not forget the additional resistance that may exist because of the breathing apparatus. Warkander et al reported that adding external breathing resistance to divers exercising at 58 m resulted in elevation of end-tidal PCO_2 ($P_{ET}CO_2$) to 72 mm Hg. At the end of the exercise $P_{ET}CO_2$ was >90 mm Hg and loss of consciousness ensued.[36] Under resting conditions in healthy individuals $P_{ET}CO_2$ is an accurate reflection of arterial PCO_2, however during exercise $P_{ET}CO_2$ tends toward mixed venous PCO_2 levels, and it thus may exceed arterial PCO_2.[37] The relationship between end-tidal and arterial PCO_2 in diving, where there may be additional factors such as V_A/Q mismatch and impaired gas diffusion, is unknown. To date there are no published data directly comparing the two values during diving exercise.

Gas Phase Diffusion Impairment

At 1 ATA intra-alveolar diffusion of CO_2 and O_2 is believed to occur sufficiently rapidly that diffusion equilibrium occurs within each breath.[38] However, diffusion within the gas phase is slowed as gas density increases and it has been speculated that during diving this might result in impairment of CO_2 and O_2 exchange, resulting in hypercapnia and hypoxaemia. The Bohr dead space is calculated using the standard formula below (Bohr equation):

$$V_D = V_T \left[1 - \frac{P_E CO_2}{P_A CO_2} \right]$$

where: V_D = dead space
V_T = tidal volume
$P_E CO_2$ = mixed expired CO_2
$P_A CO_2$ = alveolar PCO_2

The Enghoff modification of the Bohr equation is to assume that $P_A CO_2$ = arterial PCO_2.

Direct measurement of arterial blood gases during experimental dives has revealed hypercapnia, which may be due to hypoventilation[35] or reduced efficiency of pulmonary CO_2 transport as measured by an increase in dead space/tidal volume ratio (shown in Fig. 9).[1,39]

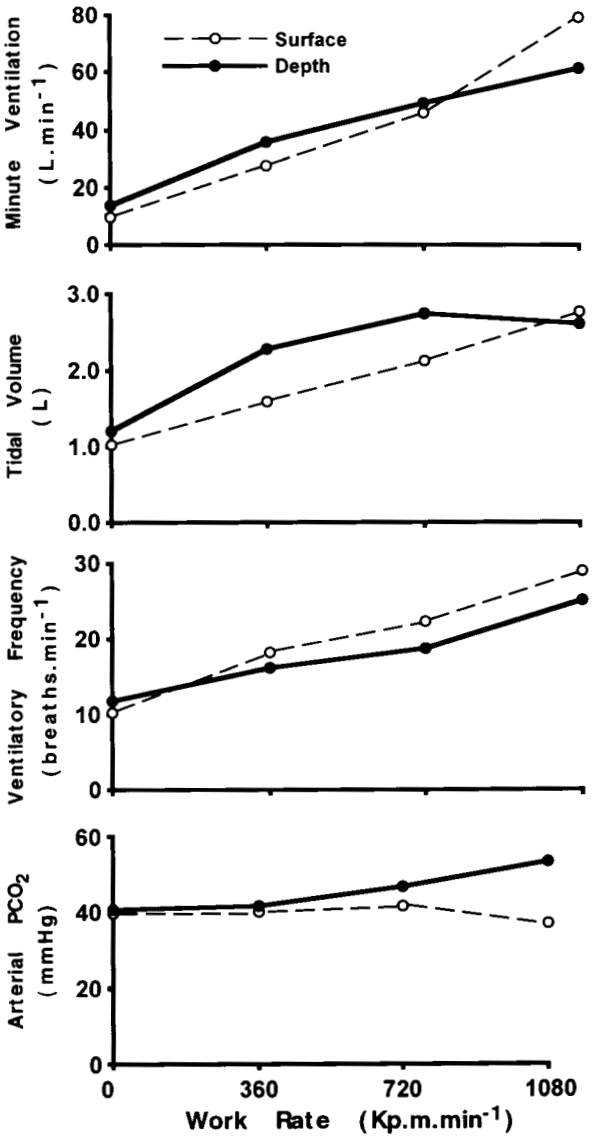

Figure 9. Exercise ventilatory response and arterial PCO_2 during bicycle exercise in a series of deep diving experiments.

Plotted data are mean values from 5 volunteers participating in experimental saturation dives to 460 and 650 m. Gas density at depth was 7.9-17.1 g/l. At rest and during moderate work loads, arterial PCO_2 at depth was maintained close to surface values, but because of greater physiological dead space a higher minute ventilation is required. At the highest work load minute ventilation approaches MVV and cannot increase further resulting in hypercapnoea. At depth the divers in this study demonstrate the typical breathing pattern of individuals with high airway resistance, higher tidal volume and lower breathing frequency. Data are from Salzano.[1]

Observations suggesting O_2 exchange impairment were made by Chouteau,[40] who noticed that goats in a chamber breathing normoxic heliox (atmospheric PO_2 = 0.22 bar or ATA) at 71-91 bar (700-900 m equivalent depth, 11-16 g/l) became ataxic and lost their footing. Increasing the chamber PO_2 reversed the situation, until, at 101 ATA (1000 m, 16.8 g/l), one animal died despite increasing the PO_2 to 0.9 ATA. Chouteau believed that this was due to impaired O_2 diffusion, and this was later referred to as the "Chouteau effect". Initially, in deep diving exposures using heliox in which humans experienced psychomotor impairment, nausea, vomiting and tremor it was suspected that hypoxia might be responsible. However, it became apparent that these symptoms, which were related to both ambient pressure and rate of compression, and became known as the high pressure nervous syndrome (HPNS), were more likely due to neuronal membrane effects and alterations in neurotransmitters unrelated to the PO_2.

Paradoxically, Gledhill observed a reduction in alveolar-arterial PO_2 gradient in subjects breathing SF_6.[41] Except in one study of three individuals, in which alveolar-arterial PO_2 difference at rest increased 2-3 fold at 300 m (heliox, PO_2 = 0.28 bar, inspired gas density 5 g/l),[42] direct measurement of arterial PO_2 in experimental dives has revealed either a reduced A-a gradient at a gas density of 3.2 g/l[39] or no significant change (up to 17 g/l).[1,35,43] Lambertsen reported a deep dive in which the ambient PO_2 in the breathing gas (up to 25.2 g/l) was maintained at 0.21 bar. Although arterial PO_2 was not measured, subjects did not report any difficulties which might have been attributable to hypoxia.[2] Actual measurement of arterial PO_2 at an inspired gas density slightly higher than in the Chouteau experiments, with an inspired PO_2 of 0.5 bar, revealed values of 200-300 mm Hg, even during exercise.[1] The Chouteau effect was therefore probably due to some phenomenon other than hypoxia, possibly HPNS. Interestingly, despite greater than adequate arterial PO_2 values, subjects in Salzano's study had significantly higher arterial lactate levels, an observation consistent with reduced O_2 delivery to exercising muscle.[1]

The issue of gas phase diffusion impairment during diving therefore remains an open one. Certainly if there is diffusion limitation to pulmonary O_2 transport it is of minor importance, at least in divers with normal lungs. Since most diving is performed using breathing mixtures with a fixed proportion of O_2, a built in safety feature during descent that will tend to offset possible problems with pulmonary O_2 exchange, is the rise in inspired PO_2 in parallel with the increase in gas density.

Although the observed elevation in Bohr dead space is consistent with gas phase diffusion limitation, there are also other explanations. An elevation in anatomic dead space due to a breathing strategy in which breathing occurs at higher lung volumes could contribute. Impaired gas distribution, causing V_A/Q mismatching, could also contribute to a higher dead space. Finally, pressure-induced dysfunction of macromolecules facilitating gas transport, such as the enzyme carbonic anhydrase, may cause arterial PCO_2 to exceed pulmonary end-capillary PCO_2 ('blood phase diffusion impairment'), thus simulating gas phase diffusion impairment and similarly elevating measured dead space.

Perhaps the ultimate experiment to assess diffusion of gases in the medium of highest conceivable gas density (1,000 g/l) was performed by Dr Joannes Kylstra. Studies in humans during therapeutic lung lavage, and one volunteer, in whom one lung was filled with saline while the other was ventilated with 100% O_2 revealed only small differences between PCO_2 values in end-tidal expired saline and arterial blood.[4,5] Given the experimental conditions of low CO_2 elimination rate and extremely slow exhalation (<3 breaths per minute) these data were consistent with complete diffusive equilibrium between alveolar liquid and end-capillary blood.

Effects of Decompression on the Lung

BAROTRAUMA

Pulmonary overpressurisation during decompression results from breath holding or bronchial obstruction and distal air trapping. The most common manifestation is mediastinal emphysema; less common are pneumothorax and gas embolism. An intrapulmonary pressure exceeding 60-80 mm Hg is sufficient to cause pulmonary damage.[44,45] This pressure differential can occur if a diver takes a full breath of compressed gas and then ascends from a depth as shallow as 1-1.5 m. AGE has indeed been reported after a dive to one metre depth[46] and in scuba divers breathing compressed air near the surface while being washed over by large waves. It has also been observed in commercial divers exposed to underwater explosions.

The numerous instances of pulmonary barotrauma (PBT) not associated with breath holding have led to hypotheses regarding regional bronchial obstruction. Dahlback and Lundgren[12] have demonstrated that immersion induces intrapulmonary gas trapping, due in large part to the increase in central blood volume.[47] Forceful exhalation during ascent from a dive might therefore generate pulmonary barotrauma. It is possible that the physiological effects of immersion may be at least in part responsible for the relatively common occurrence of pulmonary barotrauma in divers in contrast to its extreme rarity in the dry chamber environment during decompression from hyperbaric oxygen therapy.

The effects of immersion to induce gas trapping may be compounded by lung pathology. Autopsy on a submariner who died during submarine escape training

revealed obstruction of the right middle lobe due to focal bronchial obstruction from a calcified lymph node.[48] Diffuse airways obstruction due to moderately severe asthma has been associated with decompression illness,[49,50] and has traditionally been a contraindication to diving.[51] However, an international panel reached the consensus that individuals with asthma in whom pulmonary mechanics can be rendered normal (including after a provocative test) by pharmacotherapy are probably not at substantially increased risk of DCI or PBT.[52] Individuals with focal air trapping due to cysts or bullae are probably at risk of pulmonary barotrauma and AGE.[53,54]

Colebatch et al. have demonstrated that divers with a history of AGE have less distensible lungs and increased recoil pressure than control divers.[55,56] In one diver, in whom spontaneous mediastinal emphysema had occurred when performing breath hold diving, after a maximum inspiration, transpulmonary pressure exceeded 70 cm H_2O, a pressure which is close to the level demonstrated to cause pulmonary rupture. The authors speculated that stiff airways may cause stress magnification at high lung volumes (i.e. during greatest stretch). It has been demonstrated that restricting lung expansion with an abdominal binder may protect against pulmonary barotrauma.[45,57] It is therefore not the increase in pressure that produces pulmonary barotrauma, but rather the stretch. It has therefore been suggested that during decompression from a dive, breathing at either high or low lung volumes should be avoided.[58,59]

EFFECTS OF VENOUS GAS EMBOLI (VGE).

During decompression, VGE are extremely common, occurring in a large proportion of scuba divers engaged in single[60] or repetitive dives.[61] A short lived decrease in carbon monoxide transfer factor (D_LCO) and arterial PO_2 occurring in parallel with the appearance of VGE have been described after a bounce dive to 55 m.[62] Hlastala demonstrated that intravenous infusion of gas in experimental animals caused an increase in high V_A/Q gas exchange units,[63] and Ohkuda et al. demonstrated in sheep that this can result in capillary leak and pulmonary oedema.[64] High levels of VGE during decompression from a dive can also produce pulmonary oedema in humans (cardiorespiratory decompression illness or "chokes").[65]

A group in which repetitive or continuous VGE have been observed is divers decompressing from saturation dives. Indeed, several reports have demonstrated that, after decompression, saturation divers have elevated respiratory dead space and reduced DLCO to a degree that correlates with a cumulative measure of VGE.[66,67]

Effects of Inspired Gases on the Lung

The pharmacological effect of a gas is a function of its partial pressure. Therefore, gas mixtures which may not be toxic at 1 ATA can induce lung injury during diving. Oxygen at a concentration of 21%, for example, can become toxic to the lung at ambient pressures greater than 3 bar (20 m) where the PO_2 = 0.6 bar. At that pressure many hours of exposure are ordinarily required, therefore this is not an issue except during saturation diving or during the treatment of decompression illness. Pulmonary O_2 toxicity manifests as substernal burning, a reduction in vital capacity, capillary leak (Adult respiratory distress syndrome or ARDS), and if exposure does not cease, death. Provided the inspired PO_2 is reduced, pulmonary O_2 toxicity is usually completely reversible. A detailed discussion of pulmonary O_2 toxicity by Clark is suggested for more detail.[68]

Conclusions

The lung is exposed to numerous stresses while diving. The lung is the origin of arterial gas embolism, and when large amounts of venous gas embolism are present it is a target organ for decompression sickness. The lung is at risk of injury due to toxic environmental gases. Finally, a testament to the remarkable flexibility of this complex anatomic structure, is the fact that the lung is subjected to gases with properties considerably different from those of ambient air and yet is able to maintain sufficient levels of bulk gas movement and exchange of both O_2 and CO_2.

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INTERPRETATION OF GAS IN DIVING AUTOPSIES

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Key Words

Accidents, bubbles, death, investigations.

Introduction

Recent autopsy protocols for diving fatalities have emphasised the importance of the detection of gas in the body to diagnose cerebral air gas embolism (CAGE), either by erect chest X-ray, CT Scan or by dissection underwater.[1-3]

Boyle's Law states that at a constant temperature the volume of a gas is inversely proportional to the pressure. Cerebral air gas embolism occurs during an uncontrolled ascent without exhalation because the volume of the gas in the lungs expands as the ambient pressure falls, forcing gas into the pulmonary circulation and thence into the cerebral circulation.

Unfortunately, very little critical analysis has been made of the significance of intravascular gas at autopsy. Intravascular gas was detected in 12 out of 13 diving fatalities autopsied at the NSW Institute of Forensic Medicine. In 5 of the 12, the history and autopsy findings did not suggest cerebral air gas embolism. What then is the significance of gas?

Could the gas be artefactual?

Forensic pathologists have long recognised that the process of decomposition causes gas formation. Bacteria proliferate in the dead body, particularly in the blood vessels, breaking down blood and tissues and generating gas in a process of putrefaction. If decomposition was responsible for the intravascular gas then this gas should also be seen in non-diving fatalities.

Resuscitation, using endotracheal intubation, positive pressure ventilation and intravenous cannulation, can cause subcutaneous emphysema and even air emboli. Eight out of 13 of the divers were subject to vigorous resuscitation. If resuscitation was responsible for the intravascular gas then it should also be present in non-diving fatalities.

Finally, at increased pressure the body absorbs nitrogen. Normally during ascent nitrogen diffuses out of the tissues and is breathed out, part of the process of decompression. However, if death occurs at depth and the body is brought rapidly to the surface, nitrogen bubbles will evolve in blood vessels and in soft tissues and are not removed because the circulation has stopped. Decompression would appear to be capable of generating intravascular gas in diving fatalities, either during or after death.

Methods

All diving fatalities in NSW are autopsied at the NSW Institute of Forensic Medicine. In the cases presented here erect chest and abdominal x-rays were taken before autopsy. Autopsies were commenced as soon as possible after death, however there were often delays in transporting the body. The body was positioned with a block under the upper back so that the chest was the highest point. The chest was opened first taking care not to cut the superficial veins of the neck. Gas was aspirated from the heart, using a Hamilton "gastight" syringe (Hamilton Company, Reno, Nevada 89502, USA).

The inferior vena cava and portal vein were opened once the block was removed from under the body. Air aspirated was analysed by the Department of Mineral Resources, Lidcombe. The diving equipment was examined and tested by NSW Police Divers, Sydney Water Police. Where dive computers were used they were down loaded and the dive profiles recorded. Air from the tanks was also tested by the Department of Mineral Resources.

Results

Twelve of 13 diving fatalities had intravascular gas.

The time between death and post mortem varied from 8 hours to 5 days. The average time was 41 hours. Two bodies were recovered from a wreck at 51 m after being missing for 3 days. If these two cases are excluded, the average delay to post mortem was 26 hours, still a significant delay.

In all 12 of the diving fatalities gas was present in the heart, neck veins, inferior vena cava and portal/hepatic veins, often with as much as 100 ml of gas in the right

ventricle. In 4 cases where a cardiorespiratory arrest had occurred on the bottom at a depth greater than 40 m, gas was also present in peripheral vessels, in skeletal muscles and in joints.

The results in 13 unselected non-diving fatalities were quite surprising. Ten of the 13 cases showed gas on X-ray and autopsy. Of these, 5 cases had small amounts in the portal and hepatic veins and 5 cases had larger amounts in the heart, neck veins and hepatic and portal veins. The interval between death and post mortem was 5 hours to 8 days (average 35 hours).

Eight of the non-divers were not resuscitated. In this group, extensive gas was only seen in the presence of obvious decomposition, however small amounts of gas were usually present in the portal hepatic veins after 24 hours.

Five of the non-diving fatality cases had endotracheal intubation and intravenous cannulae; Three of these showed extensive gas. In 2 cases autopsied at 4 and 8 hours after death, there was gas in the heart and neck veins. The short post mortem interval suggested that the gas was not due to decomposition and suggested that resuscitation could introduce gas.

In the last 5 non-divers analysis of the intravascular gas was performed. In 3 cases the analysis was close to normal body percentages. The two cases with decompositional gas yielded quite different results (Table 1).

Discussion

Intravascular gas formation does occur in non-diving fatalities. In unresuscitated cases it appears at around 12-24 hours in the hepatic/portal venous system. This is consistent with what we understand about putrefaction as the source of the bacteria appears to be the gastro-intestinal system. The amount of gas seen is small compared to the diving fatalities unless decomposition was obvious macroscopically. Analysis of the intravascular gas shows high levels of CO_2 and hydrogen which may be the best markers of decomposition.

It appears that resuscitation can cause the early appearance of large amounts of intravascular gas. This was a surprising result. Intravascular gas may represent vigorous positive pressure ventilation in a dying patient or perhaps gas introduced by the intravenous cannulation, although in all cases the cannulae had been capped or were still attached to giving sets. Since eight of the diving fatalities underwent resuscitation I cannot exclude resuscitation as a cause for gas in some of the diving fatalities.

In five of the diving fatalities, the individual was observed unconscious on the bottom at over 40 m and the body was then brought to the surface rapidly. In two of the cases, the divers became trapped in a wreck at 51 m, ran out of air and were not recovered for 3 days. These two cases showed advanced decomposition. In two cases, drowning due to oxygen toxicity at 47 m and drowning due to using air and poorly maintained equipment at 76 m, the body was brought rapidly to the surface. X-Rays at autopsy showed extensive gas in soft tissues, muscles, joints and peripheral vessels, as well as in the heart. The gas here is most probably due to peri- or post-mortem decompression. In the 5th case, the cause of unconsciousness was not established, however, given the rapid ascent of the body from 46 m, I am unsure whether the gas seen represents a cerebral air gas embolus or post mortem decompression.

Of the remaining 7 diving fatalities, four had strong evidence of cerebral air gas embolism. Two lost consciousness suddenly on reaching the surface. In one of these the dive computer demonstrated a 20 second ascent from 30 m. In two other cases, the presence of a perforated ear drum and an empty tank suggested a rapid ascent.

One case, who had a 70% stenosis of the left anterior descending coronary artery, became unwell on the bottom and unconscious during the ascent. The presence of the gas suggests a cerebral air gas embolism during ascent even though the primary cause of death appears to be ischaemic heart disease. In the remaining 2 cases; the presence of the intravascular gas implies a cerebral air gas embolism, although there is no history of a rapid ascent.

TABLE 1

GAS ANALYSIS OF NON-DIVING AUTOPSIES SHOWING GAS ON X-RAY

Gases	Three normal bodies		Two decomposing bodies	
			Case 1	Case 2
Nitrogen	77.3%	(range 75.9 - 78.8%)	23.0%	46.0%
Oxygen	18.7%	(range 16.8 - 20.6%)	5.0%	8.0%
Carbon dioxide	4.3%	(range 3.8 - 4.5%)	45.0%	3.02%
Hydrogen			26.8%	14.0%
Methane				0.1%

Large amounts of gas were seen in the venous circulation in all of these cases. If we postulate that, in a rapid ascent,barotrauma causes expanding gas in the alveoli to leak into the pulmonary veins, then one would expect most of the gas to be in the arterial system, but this is not the case. This pattern of distribution of the gas may indicate that the lungs trap gas bubbles more effectively than other tissues, such as the brain,[4] once the gas has passed thought the systemic circulation. Alternatively the gas may enter the venous circulation through the pulmonary lymphatic system as has been suggested in neonatal systemic air embolism.[5]

Conclusions

Decomposition and resuscitation can result in intravascular gas at autopsy. In view of the possible confusion arising from the production of decompositional gas, in performing diving autopsies it is important to autopsy the body as soon as possible after death. Analysis of the gas for hydrogen and carbon dioxide may indicate whether the gas is due to decomposition or not. In deep diving fatalities some gas formation due to decompression does occur.[2,3] It is not clear whether decompression makes any contribution to intra-vascular gas formation in diving fatalities during more superficial dives.

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OPEN CHAMBER CARDIAC SURGERY: A CLINICAL INJURY MODEL FOR ARTERIAL GAS EMBOLISM

Simon Mitchell, Ora Pellett and Des Gorman

Summary

Cerebral arterial gas embolism (CAGE) can occur in divers breathing compressed gas and as a consequence of some medical procedures. Open chamber left heart surgery, in particular, is invariably complicated by CAGE, although manifestations may be "subclinical". This paper discusses the utility of open chamber left heart surgery as a clinical injury model for CAGE in divers and other groups.

Key Words

Bubbles, CAGE, cardiovascular, investigations, research.

Review

Cerebral arterial gas embolism (CAGE) may occur in compressed gas diving following either pulmonary barotrauma or right to left shunting of venous inert gas bubbles.[1] Clinically, in divers, there is rapid onset of neurological phenomena such as altered consciousness, visual changes, cognitive changes, and sensorimotor deficits. The natural history of CAGE in divers is complex and may include death (often by drowning), persistent neurological symptoms, spontaneous recovery and relapse despite initial spontaneous recovery. CAGE is one of several mechanisms of bubble-induced injury collectively referred to as decompression illness (DCI).[2] The contribution of CAGE to morbidity in many cases of DCI is difficult to assess since several injury mechanisms may be involved simultaneously. This difficulty complicates the study of therapeutic intervention targeted at CAGE and is compounded by the usual absence of any pre-morbid data, a variable extent of injury and an even greater variation in the time from onset of illness in divers to presentation for treatment.

Cerebral arterial gas embolism also occurs in cardiac surgery. Any operation supported by a cardiopulmonary bypass (CPB) circuit carries the risk of CAGE as there are numerous ways in which air can be introduced into the body from the circuit. These include: incomplete pre-bypass de-airing of the circuit;[3] the use of a bubble oxygenator;[4] low blood volume in the venous reservoir;[5] high blood flow rates and entrainment of air into the venous inflow to the CPB machine.[6] In operations where the heart is not opened (closed chamber procedures), such as coronary artery bypass grafting (CABG), the CPB circuit is the main source of emboli. However, there is much

greater risk of CAGE in those procedures involving open chamber surgery of the left heart, such as valve replacement and repair of congenital defects. Air introduced to the heart chambers during these procedures may be ejected into the aorta when the heart is closed, resuscitated and allowed to work. Surgeons attempt to remove intracardiac air before cardiac ejection using "de-airing" techniques. However, studies utilising echocardiographic or Doppler techniques have documented that conventional de-airing techniques are not completely effective.[7-12] Recent studies have suggested that these conventional techniques fail because air is trapped in the pulmonary veins during surgery[7,11] and is not dislodged until the heart is resuscitated and pulmonary blood flow increases to normal levels.[7] These bubbles will pass to the left heart and in turn into the aorta. A novel and effective de-airing technique which allows the heart to develop physiological output without ejection into the systemic circulation has been reported[13] but is not yet widely used.

The use of cardiac surgery patients in a study of CAGE pathophysiology or treatment confers the following significant advantages over a group of divers with DCI: premorbid neurological and neurocognitive function can be assessed so that each patient can serve as their own control; the exposure to bubbles can be quantified using Doppler devices; and any treatment can be given at the same time in relation to the emboli exposure. However, before cardiac surgical patients are used in this way, a relationship between bubble exposure and outcome must be demonstrated. Moreover, to allow for reasonable group sizes in a trial of different treatments, adverse outcomes must occur frequently.

There is a plethora of studies showing adverse neurocognitive outcomes in patients after cardiac surgery. Many (50-70%) cardiac surgical patients are reported as suffering from post-operative cognitive deficits of some type, and 2-5% are said to suffer a peri-operative stroke.[14] It is known that the incidence of post-operative neurocognitive deficit is higher in coronary artery bypass graft (CABG) patients than in patients undergoing forms of vascular surgery which do not involve CPB,[15] and it is generally agreed that embolism of the cerebral circulation is an important contributing factor to such deficits.[16]

The argument that embolic brain injury is common and important in closed heart cardiac surgery such as CABG is supported by studies in which any deterioration in post-operative neurocognitive performance has been correlated against emboli exposure measured using Doppler devices. Stump and colleagues[17] reported a mean left common carotid artery emboli count of 130 in patients with neurocognitive deficit 5-7 days after CABG surgery and of only 63 in patients without deficit. Pugsley and colleagues[18] reported the percentage incidence of neurocognitive deficit at 8 weeks after CABG surgery as 9, 23, 31, and 43 in patients exposed to <200, 201-500, 501-1000 and >1000 middle cerebral artery (MCA) emboli respectively. Clarke and colleagues[19] reported cerebral complications after CABG surgery in 70% of patients exposed to greater than 60 (mean 118) MCA emboli and in only 30% of patients exposed to less than 60 MCA emboli.

Although these studies support the hypothesis of embolic brain injury during surgical procedures involving CPB, attribution of these injuries solely to bubbles is not possible. Although the Doppler devices used are more likely to detect bubbles than solid emboli of the same size,[20] CPB does generate non-gaseous emboli such as antifoam particles[21] and fibrin-platelet aggregates.[22] Indeed, there is still a considerable uncertainty about the relative importance of the different emboli generated by CPB. Moody and his colleagues used an alkaline phosphatase staining technique to demonstrate small capillary and arteriolar dilatations (SCADs) in the brains of patients who died after CPB and in dogs sacrificed after experimental CPB.[23,24] No SCADs could be demonstrated in control patients or animals who had not undergone CPB. The SCADs exhibited menisci at their ends and appeared empty. They were initially postulated to be air or fat emboli,[23] but subsequent work has shown that some of these lesions exhibit granular birefringence. Others have stained positive for glycoproteins and glycolipids.[24] These would be unusual findings in bubbles, although a bubble may acquire a lipid content as it strips surfactant from the vessel endothelium.[25]

In view of this confusion, closed chamber cardiosurgical procedures in which CPB is the principal source of emboli are probably not a suitable clinical injury model for CAGE in divers and other groups primarily injured by bubbles.

A much greater degree of cerebral embolism in open chamber procedures has been documented using Doppler devices. Stump and his colleagues[26] recorded a mean of 1339 left common carotid artery emboli (range 38-4455) throughout open chamber valve replacement surgery. In contrast, closed chamber CABG patients were only exposed to a mean of 62 emboli (range 23 - 107). Other researchers have recorded similar results.[7,9] This greater exposure in valve surgery patients is almost certainly accounted for by bubbles ejected from the left heart at the end of the procedure. Studies using transoesophageal echocardiography in the investigation of de-airing techniques support this contention. Bubbles have been observed emerging from the pulmonary veins soon after heart ejection is resumed,[7] or being trapped in sites like the left ventricular apex and right upper pulmonary vein under the influence of buoyancy, only to be "stirred up and expelled away" with the resumption of cardiac ejection.[11]

Despite the greater bubble exposure in open chamber surgery patients, comparisons of neurological outcome between this group and closed chamber (CABG)

surgery patients are confusing.[27] However, such comparisons are tenuous since CABG patients are usually older and affected by other risk factors for poor outcome such as carotid artery and cerebrovascular disease. The most recent review of this issue suggests that, on balance, open chamber patients are at greater risk of adverse neurological outcome.[27]

Nevertheless, for open chamber heart surgery patients to be used as a model of CAGE, it is still important to show a correlation exists between neurological outcome and bubble exposure. Surprisingly, there are no relevant published studies. However, we have produced some relevant preliminary data from our randomised prospective double blinded trial of lignocaine in cerebral protection in open chamber left heart surgery at Green Lane Hospital, Auckland, New Zealand.

Methods

The subjects were 23 patients undergoing left heart valve replacement surgery and who were enrolled in the lignocaine trial. This trial was approved by the North Health Ethics Committee in August 1994. There were 15 males and 8 females, and the mean age was 54.9 (SD 10.1). All patients underwent a battery of 11 neurocognitive tests before surgery. All tests were performed by the same research psychologist and in most cases were conducted within the same designated office at the hospital.

The right common carotid artery was continuously monitored for emboli activity from 5 minutes before cardiac cannulation until 20 minutes after withdrawal of CPB, using a Rimed Flowlink 300 colour flow Doppler (Rimed, Tel Aviv, Israel) interfaced to a purpose built emboli counting microprocessor. The nature and use of this device has been described in more detail elsewhere.[5] Doppler signal processors may be confounded in many ways,[28] and emboli "counts" are not exact. We therefore refer to the count as an "index of microembolic activity" (IMA). Nevertheless, calibration of our device has shown that increases in IMA are directly proportional to emboli numbers.[5]

The neurocognitive tests were repeated for all patients at 6 to 8 days, eight weeks and six months after surgery. Once again, all tests were conducted by the same research psychologist. Both the patients and the psychologist were blinded to the emboli exposure.

Only the change between the pre-operative and eight day performance in a computer generated test of reaction time (psychomotor speed) was analysed before the 1997 SPUMS Annual Scientific Meeting. A univariate linear regression was performed for the relationship between emboli exposure and any change in reaction time at eight days after surgery using the Systat statistical package.

Results

IMA ranged from 247 to 6,959 with a mean of 2,334 (SD 1694) for the 23 patients. Most emboli (mean fraction of total = 0.73 (SD 0.26) were recorded at the end of the procedure when the heart began to eject. Almost all signals were ultrasonically typical of bubbles. The regression plot for the relationship between IMA and change in reaction time at 8 days post surgery is shown in Figure 1. Note that an increase in reaction time (indicating a in performance) is plotted as a *negative* value. There was a significant decline in performance as IMA increased ($p < 0.025$).

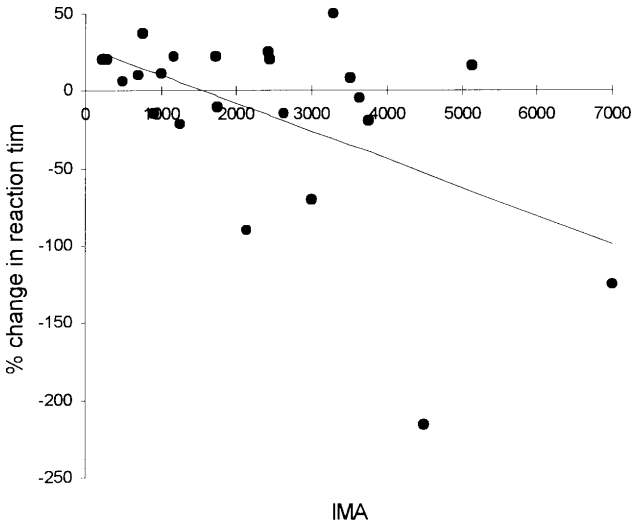

Figure 1. Relationship between change in reaction time at 8 days after surgery and perioperative emboli exposure An increase in reaction time is plotted as a negative value. R=0.52. p<0.025.

Discussion

These preliminary results suggest that increased exposure to emboli in open chamber left heart surgery is associated with a decline in neurocognitive performance. Given that the vast majority of the emboli are bubbles, this finding would support the use of open chamber cardiac surgery as a clinical injury model for CAGE in divers and other groups. However, this preliminary data must be interpreted with caution. First, all patients were part of a randomised, double blinded trial of lignocaine in cerebral protection during cardiac surgery which was not unblinded for this analysis. It follows that approximately half of these patients have received a drug which may have modified their neurocognitive outcome. Second, this analysis describes outcome in 1 of 11 tests, at 1 of 3 points in time, for 23 of 60 patients. We must wait for completion of all patient follow up, for unblinding of the lignocaine trial and the analysis of results for all tests at each follow up before drawing firm conclusions. This data will be reported in due course.

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REPRINTED FROM THE ABSTRACTS OF THE 1997 SCIENTIFIC MEETING OF THE UNDERSEA AND HYPERBARIC MEDICAL SOCIETY

A superior heart de-airing technique reduces cerebral arterial gas embolism following left heart valve surgery. Milson FP and Mitchell SJ. *Undersea Hyperbaric Med* 1997; 24 (Suppl): 24

Abstract

Background

Cerebral arterial gas embolism (CAGE) invariably occurs at termination of cardiopulmonary bypass (CPB) in left heart valve surgery. CAGE is not prevented by conventional left heart "de-airing" methods and has been linked to post-operative stroke and neurocognitive deficits. We have developed and assessed a novel de-airing technique which utilises high flow left ventricular and aortic venting from the working heart into the CPB venous line, prior to aortic declamping. This technique allows the heart to establish physiological output before the aortic clamp is removed. Ejected emboli pass to the CPB circuit, rather than the systemic circulation, and are removed by the circuit's protective elements.

Methods

The right common carotid artery was monitored throughout the left heart valve surgery using a colour flow Doppler interfaced to an emboli counting microprocessor in 21 consecutive patients de-aired conventionally (group 1), 9 consecutive patients de-aired by the novel technique (group 2) and 4 coronary artery bypass graft patients who did not require de-airing (group 3).

Results

The mean emboli count recorded after aortic declamping was 2,580 ± 321 (SEM) and 293 ± 110 in the group 1 and group 2 patients respectively (p=<0.001). The efficacy of the novel technique improved during the series: four of the last five group 2 patients were exposed to similar trivial numbers of emboli after aortic declamping (less than 20) as the group 3 patients.

Conclusions

Cerebral arterial gas embolism associated with left heart valve surgery is significantly reduced by the novel de-airing technique.

From

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Key Words

Bubbles, CAGE, cardiovascular, investigations, research.

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

FORMIDABLE!

Colin Taylor

More than 100 years before Jacques Cousteau claims to have invented scuba diving, a Paris dentist beat him to it. A French diving historian has produced the drawings, shown here, from a patent application for equipment successfully used for diving in 1828!

Ask who invented the first self-contained underwater breathing apparatus and you will probably hear the names of Cousteau or Hans Hass. Unless, that is, you go to St Brieuc, in Brittany, and speak to Daniel David, a keen diving historian, who has uncovered the patent documents for what is probably one of the first scuba sets invented, with all the ingredients which make up a modern day set of equipment at that!

The rig, shown in drawings completed by Daniel (Figure 1 on page 116) from the written description and drawings in the patent records (Figure 2), was dived successfully in 1828. It seems incredible that the knowledge of it was completely forgotten for 150 years.

Daniel was carrying out research into a wreck called the *Bellona* when he heard in the coastal towns of northern France the name of D'Augerville, who had tried to salvage the contents of the ship using a diving apparatus he had designed. After several years of research, Daniel discovered that the inventor was not an engineer, salvage expert or diver, but a dentist from Paris called Lemaire D'Augerville. He actually lived in the street where Cousteau now has his headquarters: Rue St Honore!

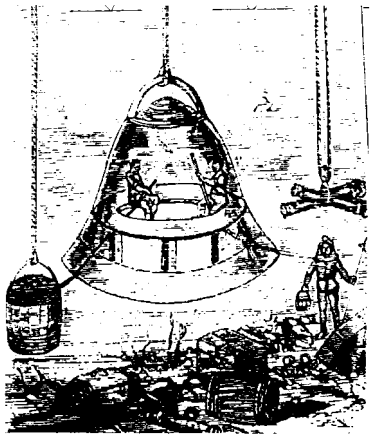

Figure 3. Typical diving bell operation of the day.

Little is known about this imaginative and clever inventor, other than that he decided to try and make some money by diving on sunken ships for their contents. At that time, dives were being carried out with the use of diving bells. Helmets or hoods would be used, linked to the bell by leather hoses. Air was replenished by sending down barrels of fresh air and releasing it inside the bell (Figure 3).

The Deane brothers were diving using their half dress with adapted smoke helmets at this time, working with Augustus Siebe, who developed the standard full dress still

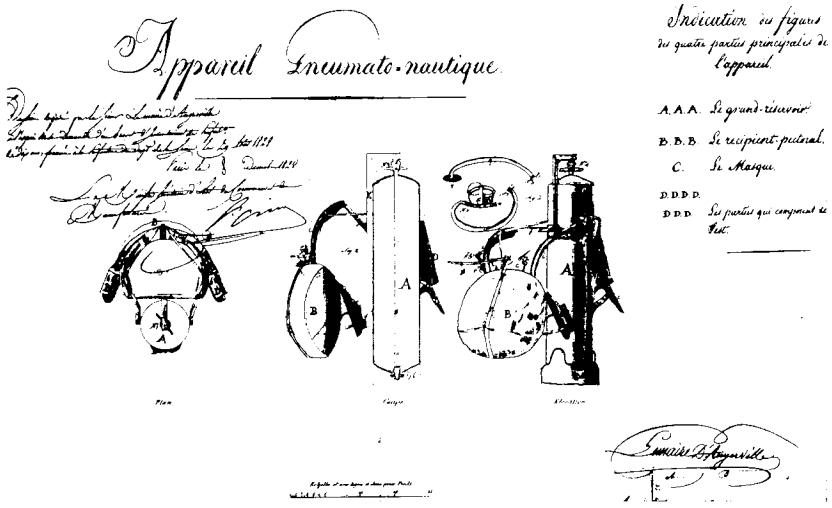

Figure 2. An extract from the original application made by D'Augerville to the Paris Patent Office for 'Appareil Pneumato-nautique".

Figure 1. Drawings, by Daniel David, of the likely appearance of the self contained underwater breathing apparatus patented in Paris by Lemaire D'Augerville in 1828.

used today. To the diving pioneers of the time Lemaire's kit must have looked space age in the extreme.

The dress consisted of a drysuit made of a type of rubberised canvas, with a lining of soft flannel ("to be warmer") which was sealed at the wrists and ankles, complete with hood. It was designed to be close fitting. It must have looked rather like the early rubber suits of the 1950s. The mask was almost a full-face mask, made of copper, sealing against the face with a soft dental cement of plasticine consistency "which could be adapted for any shape of face" and enclosing the nose to prevent mask squeeze.

The cylinder was a low-pressure cylinder pumped by hand to not more than 23 bar, the limit for the pumps built at that time, and much larger than its modern equivalent at about 1 m long, with a diameter of 150 mm. This was attached to the diver's back with straps.

Air was supplied to the diver via a tube, and flowed constantly through a lung attached to the chest. As the diver went deeper, he would operate a valve to give himself more air. As you can see from the drawings (Figure 1), the rig was even fitted with an independent ABLJ, with its own emergency cylinder, equipped with both an inflation valve and a dump "for ascending and descending".

The weight belt was rigged in such a way that it could be dumped very quickly by operating a lever at chest level. If the diver ran out of air, he merely dumped his weights, put his finger in the exhaust valve and breathed on the remaining air left in the lung until he got to the surface. As he would only have been diving in shallow water, this worked very well, with no known accidents.

Divers successfully used this outfit over a number of years to dive to 6-9 m for up to 30 minutes and reached depths of 20 m on occasions. The only item missing was a pair of fins, which really would have made the rig look like a modern-day copy.

D'Augerville intended his diver to swim, and described how to obtain negative buoyancy by adjusting the air in the ABLJ. The rig was fairly heavy, weighing in at 50 kg, but the diver would have been able to walk or pull himself around. A compass in a glass dome was even fitted to the front of the rig on the weight dump lever.

The equipment was extremely sophisticated for its time, and D'Augerville shows an amazing knowledge of the scientific principles he employed. Whether he was self taught or had help from someone else we shall probably never know. If it was his own creation, then he was a genius indeed, and clearly missed his vocation.

Unfortunately for Lemaire D'Augerville, his invention never caught on, and faded into obscurity, probably because of its limited depth range and use. Maybe it was just too futuristic for the divers of that era.

Daniel David is continuing with his research, and has made some exciting discoveries. He is gathering material for a book, and is an active member of the Historical Diving Society, seen exhibiting at recent dive shows and exhibitions. He continues with his research all over France. His revelations will no doubt upset one or two people who have claimed to be first with innovations which have made scuba diving possible.

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THE SPONGE DIVERS OF SYMI

Frank Allen

The small Greek island of Symi, in the south-eastern Aegean, was once home to some of the world's finest sponge divers. Such was the value of their harvest that the island became one of the wealthiest seaports in the Mediterranean. Today, little of this is evident at first glance. Despite the exceptionally clear water and steep drop-offs that make this the perfect diver's island, there are no diving schools or facilities. Strict laws prohibit sport diving in an effort to protect the many archaeological treasures still lying off the island's shores. Visitors may venture under water only in snorkelling gear, to glimpse the island's steep blue slopes in the fleeting seconds of a held breath. Today this rugged, steep sided island is sought out by those with a taste for tranquillity and the simple things in life, but to explore the narrow streets of the island's town, Yalou, is to turn a page of diving history.

Imagine, if you can, sliding down an inclined plank from a boat into the sea, clinging firmly to a large, flat stone. You plummet vertically, clearing your ears against a primitive nose-clip while trying to judge the approach of the bottom a difficult task, as you have no mask. Using the rock as a crude hydroplane, you angle it upwards to avoid a

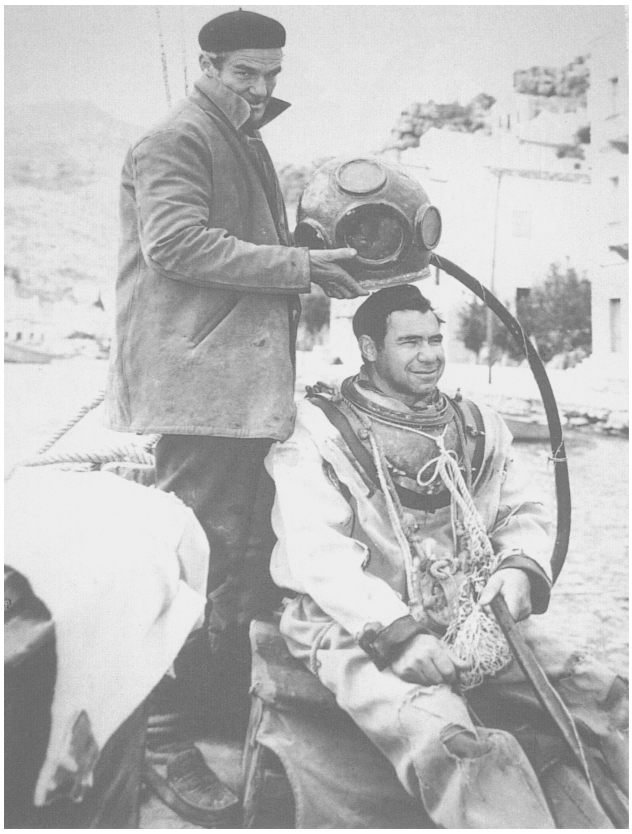

Figure 1. A sponge diver suiting up in the '50s.

high-speed crash, and so arrive on the seabed to begin work. The depth is 20 m and your task is to locate and cut free as many sponges as you can before your breath runs out. This is a job made doubly difficult by the fact that you have no fins.

Your only luxury is a light line tied around your wrist. One sharp tug will get you a free ride back to the surface. After a short rest, down you go again.

This was the ancient way of sponge-diving, or "naked diving". It is a technique beautifully depicted on a has-relief in Yalou' s small museum. It was the method used by local hero Stathis Hatzis in July 1913, when he secured a line to the fouled anchor chain of an Italian battleship off the island of Karpathos. Reaching a depth of 88 m in an incredible dive lasting more than three and a half minutes, he was rewarded with a gold medal and the right to travel free for life on any Italian ship of his choice.

But it was not Symi's naked divers who brought fame and fortune to the island. It was the arrival of technology. At some time during the middle of the last century, a set of Augustus Siebe's revolutionary diving gear turned up on the island and transformed its primitive sponge diving industry at a stroke. By using the distinctive copper helmet and canvas suit, divers could not only see clearly underwater for the first time, but could venture deeper and for longer than ever before.

The introduction of this equipment heralded a "gold rush" for sponges. By 1880, fortunes were being made from the rich harvests gathered by the island's first generation of technical divers. Much of this money was reinvested in the industry to provide more boats, crews, and divers. Waterfront properties were converted into processing plants, where whole families were employed to beat, clean, clip and pack the precious sponges for export to every major city in the world.

Soon almost every islander was involved in the sponge trade, working at sea or finding employment in the supporting enterprises ashore. By now, the building trade was trying to keep pace with increasing demands for new villas, which took shape high on the hillsides overlooking the harbour. They were more spacious and opulent than anything previously seen on Symi.

In the town's only foundry, the original Siebe diving dress and pump were copied and reproduced by Symiot craftsmen, using basic blacksmith's tools and a forge. As a result, most diving was being done with locally made gear, which could be replaced or repaired on the spot.

As the most accessible sponge beds were gradually depleted, the divers were forced to venture deeper and further afield. Working in the indigo twilight beyond 60 m, with no knowledge of decompression illness or dive tables, their working lives were short.

Many would survive deep diving over a long period, only to be killed or crippled for life when narcosis so muddled their minds that they were unable to answer simple signals on their lifelines.

Believing their divers to be in the clutches of some unknown horror far below, the linesmen would pluck them from the seabed and haul them back to the surface as fast as muscular arms could work. In such situations, most divers would die purple-faced and broken, at the feet of men whose only thought had been to save their lives.

By 1910, the sponge fleet was spending up to seven months a year away from home searching for new grounds and its passage was marked by a sad trail of divers' graves. Divers continued to push the limits in leaky suits and with foundry-made pumps barely able to supply enough air to keep them alive.

One man in three was either dead, crippled, or marked for death before he reached marriageable age. For the families waiting at home, sponge diving became known as The Tyranny. It took away husbands, fathers and sons, and left behind a community to carry on as best it could.

The situation could not be sustained, and eventually the Symiots gave up diving altogether. By 1919, the boats leaving Symi were still manned by Symiots, but they carried volunteer divers from the nearby island of Kalymnos,

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Figure 2. A pump, helmet, hose and breastweight: relics of sponge diving in Symi a hundred years ago.

who became the ones to risk their lives. The Symiots risked only their money.

With the later development of man-made sponges, the industry gradually spluttered to a halt, and by the 1950s it had faded into history.

Today, the gentle mask of tourism hides much of this story, but the inquisitive visitor can still find evidence of Symi's diving past. Old copper helmets, ancient pumps and even the old foundry can be found among the sleepy streets of the town. Along the quay, a sprawling boat yard still marks the spot where hundreds of sponge-boats first took to the water. On the hillsides many crumbling villas, once the pride of wealthy sponge merchants, maintain a forlorn vigil across the harbour.

Perhaps the most touching of all links with the past is the way it is remembered in dance. The Bends Dance, performed in Symi to this day, is a depiction of vigorous youth reduced to shuffling helplessness by the voracious demands of The Tyranny. It is a fitting reminder of what was once the true price of a sponge.

Meanwhile, beneath the sea, sponges are gradually making a comeback. They are not yet plentiful, but they can be found readily enough by a fit snorkeller and can be collected, as in ancient times, on a held breath at depth.

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