South pacific underwater medicine society

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EDITORIAL

There are two matters of major interest to all divers, even those least interested in the theory of diving. These are decompression sickness and how to prepare for the possibility of running out of air at a rather inconvenient time, such as when submerged at depth. Both these matters receive attention in this issue, though naturally neither subject's problems can be considered to be entirely resolved thereby. As Dr Bronowski so ably stated the matter, "There is no absolute knowledge. And those who claim it, whether they are scientists or dogmatists, open the door to tragedy. All information is imperfect. We have to treat it with humility. That is the human condition." Nevertheless it is hoped that the fog of disputation will thin a little after readers have given due thought to the contents of this issue. All too often discussion of these matters causes one to recollect FitzGerald's famous translation of Omar Khayyam:

Myself when young did eagerly frequent Doctor and Saint, and heard great Argument About it and about: but evermore Came out by the same door as in I went.

It is hoped that readers will be left with a better appreciation of the complexities of diving problems, including the paucity of information from which many cherished beliefs originate. Although many things are "self evident", few are in truth decided for all time. Your comments and suggestions on these, and any other matters, are welcomed. The pages are open to all to share opinions, information, conclusions, and gossip. And how the more welcome if you can share a paper ready for publication!

In the matter of the "Free Ascent" controversy we have a fine range of contributors, each with a personal viewpoint. There is, remarkably, less division of opinion nowadays than formerly, though the reasons are probably a compound of fears for the Instructor's (Legal Liability) safety more than out of any clear thought and wholesale conversion to a One True Faith concerning safer diving. Information extracted, legitimately or otherwise, from Naval sources relating to SETT experience seems to indicate that true "free ascent" is the most dangerous method of training for an emergency ascent, it being far less dangerous to ascend with a mouthpiece available and some buoyancy assistance. It is to be noted here, as in the whole gamut of Medicine, cases will range in severity from those barely detectable except by the employment of special apparatus to cases where death unmistakably attests to the existence of some pathology. It is perhaps sobering to realise that even compression/ decompression in a Chamber can result in Air Embolism. Dr Harpur has applied knowledge of the possibility (certainty?) of airways closing during the exhalation of ascent to the old information that an "empty" tank may yet have a useful breath or two left in it as the surface is neared. It is now the fashion for Instructors in many organisations to treat ascent as something requiring special teaching attention and to have their pupils practice out-of-air ascending up fixed lines while wearing full equipment and with the mouthpiece held in the hand. This, though hardly a simulation of an Emergency situation, seems to be a reasonable compromise position at the present time. The RAN seem to have worked out a near compromise in this training but not one that is available to the common run of divers. It will be interesting to read case reports of any oxygen embolism incidents that may be recognised in RAN personnel over the coming years.

One area where more information is required is that relating to the reasons why divers need to make emergency ascents. The pilot study undertaken and reported by Doug Walker indicated that a more widespread and intensive approach to divers could turn up extremely useful information. It is hoped that readers will contact him on this matter. After all, there may exist a possibility that a change in basic training, equipment design or diving procedures, or even the minimal equipment a careful diver would always have, could be suggested were our information more accurate. The vest out-of-air, or other, Emergency is the one that never develops. Mr Peter Harrigan, our Honorary Cartoonist, has caught the essence of the dispute over the training dilemma Instructors face and we are again grateful to him for his contribution to both our amusement and our enlightenment.

We are honoured to have an article from Professor Hills. He is a world renowned expert on decompression matters and even those who tend to get a glazed look when faced with formulae from the metaphysical reaches of mathematical medicine, like your Editor, can gain much from reading him. And the Check List for possible cases of DS is to help recognise the critter if it does occur!

Commander Warner has presented a paper for comments and these can be sent either via the Editor or directly to him at the Department of Energy (Petroleum Engineering Division), Thames House South, Millbank, London SWIP 4QJ, United Kingdom). It is hoped that someone will make the effort to reply, for the sharing of thought and experience between different areas of the world will improve safety. Safety is, of course, Commander Warner's main concern and this report of his 1978 talk in the USA demonstrates his concern. The Melbourne meeting similarly dealt with safety, demonstrating the complexity of the problems as well as the interest in overcoming them.

There seems to have been a sudden upsurge of active interest in the problems women divers face and a desire to determine the actual troubles specific to their sex that have occurred. Till now advice to women has been based on theoretical considerations, an excellent basis only when the alternative is a blind guess. The American women divers are to be commended in their present active attempts to obtain information directly from those involved. A recent NAUI survey was sent personally to 20 of our women members , and 9 replies were elicited. This compares favourably with an apparently nil response to a survey on Octopus rigs distributed to some members of an Instructor organisation at the same time. The papers taken from the IQ9 meeting pre-date this survey and a report on the results will be published when available. In this and other matters we are indebted to both the writers and to NAUI for permission to reprint their papers.

On a less formal level, it is hoped that the minor items provide both amusement and at least a momentary pause while their relevance to diving is savoured. And finally a footnote from our cartoonist, a contribution beyond the normal call of duty! Knowing that members, and even your Editor sometimes go on holidays naturally <u>always</u> in association with their work, he has added his ideas on their possible choice of travel arrangements.

Perhaps we don't deserve it, but thanks all the same!

Ban Voyage Petertange

DR VICTOR BRAND WRITES:

I must thank John Archdeacon for making an attempt at solving the problem which I posed in an earlier number of the Newsletter. He has arrived at an ingenious explanation which however, does not fit the facts.

Firstly, Laryngeal Spasm shows a characteristic picture of upper airway difficulty with "crowing". I'm quite sure that it was a case of deeper obstruction - most probably Bronchiolar constriction.

John ignores the most important and serious fact that the diver lost consciousness, this together with the respiratory difficulty can only indicate Barotrauma and probably air embolism.

The explosive onset of the condition is surely quite understandable. Visualize a section of lung expanding to the limit of its elasticity and then bursting. I will cite an example to illustrate this from my store of rancid reminiscences.

The diagnosis of Psychotrauma, by which I presume he means panic, does not result in acute syncope - the reverse is most likely.

I agree that it is a pity that the equipment was not checked. The incident occurred in a rather undeveloped area on the Gulf of Aquaba near Eilat, and the tank and regulator (a Poseidon) was mixed up with other gear while the diver's head was clearing.

I would like to know whether this sequence of events has ever been known to occur in any equipment. John first supposes second stage failure and then later on first stage failure!

The incident that I would like to cite as an example of the explosive nature of barotrauma occurred about 18 years ago when I was diving with a group at Heron Island. We dived to the bottom of the Wistari Channel about 120 feet (contents gauges were not de rigeur in those days). One diver ran out of air on the bottom and his reserve valve jammed. He went up with the diving guide buddy breathing and I with them.

All was going very well until at about 20 or 15 feet where Boyle's Law showed its teeth - the diver coughed explosively and a big brown cloud spread around us. Luckily the victim had no sequelae to this burst lung and was diving again after 24 hours.

* * * * * * * * * * * * * *

HOW ANEMONEFISH SURVIVE SEA ANEMONE NEMATOCYSTS

Doug Wallin has reported (<u>Sea Frontiers</u>, 24(1), 1978) recent studies of this surprising survival of anemonefish in its chosen habitat. The mucus covering the skin of each fish contains an inhibitor chemical that prevents the nematocysts from discharging. The fish acquire this ability after birth, lacking this immunity when first settled from the plankton. This takes about an hour, during which time it repeatedly brushes briefly against the tentacles. The anemone tentacles themselves must obviously contain a similar chemical to avoid stinging each other into impotence.

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Emergency Ascents: some background information Dr Douglas Walker

A man soon dies unless supplied with adequate oxygen and purged of excess carbon dioxide and anyone so placed that his respiratory requirements can be interrupted at any moment has a constant awareness, to a greater or lesser degree, of the need for urgent action should such a problem arise. The most obvious action would be to rush for the open air, an option that may be difficult to put into practice in the case of a diver underwater.

Such a person has not only a distance to travel to reach the surface, which takes time he can ill afford, but also a pressure differential to traverse that can prove fatal under certain circumstances. Unless an alternative source of air can be obtained rapidly he will be faced with a choice between the possibility of suffering a "burst lung" during an emergency out-of-air ascent and the certainty of drowning should he remain underwater. Should panic intervene the victim will be unable to make any rational decision and forfeits his chances of survival, therefore training must be designed both to reduce the chances of such situations occurring and to inculcate a planned reaction so thoroughly that it will blank out at least the early stages of the panic response. Controversy exists, however, concerning the form such Some hold that everyone should actually perform one, or training should take. several, out-of-air ascents during the "cold" non-panic situation of the initial (or later) diver training sessions that precede each phase of diving fitness certification. The intent is to let every diver discover for himself the practicality of such ascents. Others believe that the possible risks associated with such ascents are unjustified, that thorough training and correct diving discipline will make such out-of-air situations extremely rare, and that one or two practice ascents do not prepare the diver for the conditions of a for-real situation. Dr Glen Egstrom has produced "learning curves" to demonstrate that it required 17-21 trials before one reaches a learning plateau, the "overlearnt" stage where behaviour is reliably reproduced without the need for conscious thought. Such a stage is hardly ever reached by novice divers, even for the basic skills of diving and would not be attainable by them re emergency ascents except at the cost of very many practice ascents. There is a lack of documented evidence concerning the proven need for, or benefit from, such training. The BS-AC has for years prohibited it as a part of their tests for certification, while the French have regarded it as essential for all divers to demonstrate "Free Ascent". There are, regrettably, no Incident Reports from France so the true safety of their methods must remain conjectural, but the BS-AC. Incident Reports seem to illustrate the small part an ability to ascend in an emergency depends on previous practical training. All parties agree, however, on the need for a thorough teaching of the theory of safe ascents to all divers at an early stage in instruction.

In addition to those who hold strong views on this matter there are many who display a fine balance of indecisiveness, debating the terminology of the various ascent procedures without examination of the basic facts. There is a further group who await an Official Verdict, aware of the balance that exists between the advantages and disadvantages of each teaching routine. It is to these the following is primarily directed.

For convenience the presentation of evidence is in three sections :

- 1. The opinions and experience of Australian diving instructors who responded to a pilot survey in 1975 concerning Emergency Ascents.
- 2. Information available from SETT and Chamber incident reports.
- 3. Information available from published reports of Pulmonary Barotrauma associated with diving.

1. Australian "Emergency Ascent" survey

In 1975 a questionnaire was distributed to members of two groups of diving instructors. This was a pilot survey intended to determine the feasibility of such an approach, divers being notoriously reluctant to put pen to paper, though not averse to verbal commentaries about diving misadventures under informal conditions. They were asked whether they believed it enough to teach but not practice Emergency Ascent (the type of ascent was not strictly defined), what their present practice was and whether they would wish to alter this if free to do so, and what occasions of "real" emergency ascent had either occurred to them or were known to them. There were 32 replies, which was sufficient to indicate the value of this type of investigation. Attempts were made to interest overseas groups in a similar project, unfortunately without eliciting much enthusiasm.

As had been anticipated there was a majority in favour of practicing of Emergency Ascent by pupils, but many qualified their opinions in a significant manner. Some of these comments are shown in Appendix A. It was noted that the term "Free Ascent" (FA) was being interpreted in a wide spectrum of ways. Some required a ditch-andascent, others carefully accompanied their pupils as they ascended with full equipment, demand valve mouthpiece in hand ready for immediate use. Some thought the training should be postponed till the diver was sufficiently experienced to seek a 2nd Class Certification, and some reserved this test for certification of instructors themselves. As it is known that the highest fatality rate occurs among the untrained or newly trained group of divers it is somewhat Delphic to state that such practice is "essential, but <u>not</u> desirable to inflict on students in their initial training". The figures were 23 (72%) in favour of practice, 9 (28%) against. The reason for opposing such practicing for emergencies was fear of a fatality occurring.

Concerning "for real" emergency ascents, seven claimed that not only had they personally never needed to take such action but they had no real knowledge of others taking it. This was in great contrast with some others who regarded an out-of-air situation as a normal occurrence experienced by most of their pupils, the instructors being so used to buddy breathing with the pupils for this reason that they regarded it as a non-event. Excluding such in-training events there were 64 incidents reported (Table 1 and Appendix A). Though the accuracy of stated depths and causes cannot be assessed and these must represent but a small fraction of the emergency ascents that occur, it is likely that the statements indicate the experience and beliefs of this group of instructors. No information was supplied as to the training, if any, these divers had received. Four cases of possible lung overpressure, non serious, were mentioned. One was a blackout near to the surface after running out of air at 100 feet at Mt Gambier luckily the buddy had an octopus rig. One diver experienced chest pain following a rapid involuntary ascent from 10 feet, the result of dropping his weight belt while instructing pupils in rough low visibility water. The third case was of subcutaneous emphysema following a hurried ascent from 20 feet when regulator trouble occurred, while the fourth was a blackout associated with an ascent from 150 feet. It is unlikely that these are the only cases that occurred, given the methodology of this survey. Pulmonary barotrauma is possibly both much more common and more benign than is generally stated, though always potentially dangerous through entry of air into the circulation.

Attention should possibly be directed strongly to the alleged causes of the out-ofair situation developing. The term "regulator failure" may in reality indicate an empty tank if the low-air situation has not been recognised, or it may indicate the need for an urgent investigation to identify the trouble accurately, "Reserve" failures are best avoided by the discontinuance of this type of unit, the use of a tank contents gauge and possibly also a sonic warning of low-air would meet requirements of safety. Hookah failures are a matter worth special consideration, if only because so many seem to think no training is necessary before they are used. The occurrence of simply out-of-air situations in the absence of equipment malfunction can be regarded as indicating bad diving discipline, though the answers of several instructors suggest an easy acceptance of this type of diving. That so many cases occur at Mt Gambier may indicate an excess of "cowboys" among occasional visitors to this area. Their survival indicates that the "terms of trade" seem to favour survival under emergency no-air ascents.

Those responding to this survey were not necessarily fully representative of the opinions and experience of all instructors but they do at least illustrate the problem as viewed by non-medical but safety orientated and active diving instructors.

2. <u>SETT and Chamber reports</u> (see also Appendix B)

There would probably be no opposition to the practicing of "Free Ascent" (FA) by all pupils under initial scuba training were it not for the strong and repeated warnings issued to civil diving groups by personnel of both the US Navy and Royal Navy. This has been due to their experience during the training of submarine crew in Submarine Escape Training Tank (SETT) ascents where deaths have unexpectedly occurred in carefully supervised physically fit men apparently making faultless exhalations. There have even been Air Embolism cases among such fit personnel undergoing Chamber pressure tests to 100-112 feet prior to the in-water training (USA, UK, South Africa). Though many question the relevance of SETT experience to the diving situation, because the ascent made is different and the subjects lack the motivation of pupil divers towards being in the water, the point at issue here is that carefully supervised ascents in carefully checked healthy young men in warm, well lit water and with emergency recompression facilities a few seconds away from the point of emergence may nevertheless prove fatal. The deaths may be insignificant statistically but are not unimportant to the victim and the relatives. Moses gives an apparent incidence of extra alveolar air and/or air embolism as 1 per 7,200 ascents (all types). His figures are subject to error as the records had not been kept in a manner designed to furnish such details, though the New London Tank figures were available.

The figures offered of morbidity refer to clinical cases and there is now evidence that many less apparent lesions are occurring. Ingvar et al. in Sweden demonstrated the occurrence of asymptomatic EEG changes after supervised SETT ascents and James in the USA has shown the presence of extra alveolar air in 2 of 170 consecutive trainees, each apparently making three ascents. This is a risk rate of 1 case per 255 ascents. While it must be stressed that these people were not clinically disturbed to any significant degree by the changes noted it may also be noted that they would not have been included in the conventional listing of morbidity following training ascents, yet would have been at risk of an air bubble reaching a vital area of the brain. Such a risk may be justified by the benefits of such training, but such benefits require to be proven first if any less stringent management of civil ascents was proposed than that followed by US and Royal Navies. It is possibly of interest that the first ever necessitous escape from a submarine, that of the three man crew of the "Sea Diver" in 1851, was totally successful from 60 feet despite an absence of training for such an eventuality. And the RN investigation of successful WW2 submarine escapes (all nations) showed that a large proportion were made without equipment or prior training. Necessity is certainly a convincing teacher.

The available figures indicate certain additional conclusions can be drawn as to the relative risks of the various ascent modes (Table 2). Under training conditions a "Free Ascent" is the most dangerous, a buoyant ascent less so, and one using a Submarine Escape Apparatus (SEA) the safest. A correctly used SEA should be as safe as a rapid scuba ascent but the use of the apparatus is disconcerting to some at the

SETT and apparently often unsuccessful under the stress of a subsunk situation.

3. Diving Training and other cases of Pulmonary Barotrauma

It is generally believed that there are few, if any, fatalities or clinical incidents associated with ascent training and that this illustrates the basic safety of the procedure. Setting aside the fallacy that nil reports indicate nil cases, there have been numerous case reports published. These cases (Appendix C) have occurred in depths as little as 8 feet. Many have not been fatal but to be reported at all they must have been significant, so it is highly probable that many less severe cases have remained unreported. In the Australian "Stickybeak" diving fatalities survey there have been four scuba dive deaths where air embolism has been a probable cause, one being a FA training ascent several years ago. Even apparently uneventful diving may be followed by evidence of pulmonary barotrauma. The US Navy has even reported an air embolism in a snorkel diver ascending from 30 feet. As an example of the need to give restricted credence to nil reports a case of a fatal practice (training) 100 feet FA is known (overseas) where not only was there neither police investigation or Inquest into the event but those involved called a person who reported the case to a friend an (expletive-deleted) troublesome fellow. The official cause of death was drowning.

It is hoped that readers will supply the author with details of incidents associated with training or emergency conditions known to them, for the presently available reports may represent a biased sample of diving incidents.

Discussion

There are several points of interest that emerge from the available facts. First, cases of pulmonary barotrauma can occur during normal diving and are of clinical significance in a statistically relevant proportion of ascents of an "irregular" nature, however thorough the precautions. Second, extra alveolar air and cerebral air embolism, the result of pulmonary barotrauma of ascent, have effects ranging from immediate death to damage discoverable only by the use of special tests (Chest X-Ray; EEG). There does not seem to be a reason why some become victims and others suffer nil ill effects while undertaking similar ascents, but the work by Walder and others suggests that in man, as in guinea-pigs, bronchospasm or bronchial obstruction by mucus may have occurred. He quotes a fatality that occurred during decompression of a man who had been effected by fumes. Thirdly, various methods of "irregular ascent" <u>are</u> at present being performed by many pupils under initial training courses. There is also probably, a significant degree of poor diving occurring if measured by the occurrence of out-of-air incidents glimpsed at in the survey.

It is of relevance to note that the only BS-AC. specific investigation for making Emergency Ascents (Hume Wallace, Kingston Branch 1956-1961) indicated that the failure of inferior demand valves then entering the UK was the commonest cause, with poor diving discipline also significant. Since <u>1966</u> the BS-AC has prohibited practice of "free ascents" and they have not been required for certification. Incident Reports since then have shown no need to change this rule. The recent introduction of Deep Rescue training has itself produced casualties, a practical example of the need to ensure that training for safety is itself safe. PRIMUM NON NOCERE should ever be our guide.

Naval experience indicates that it is safest to ascend with buoyancy and with some source of air, conditions best met with the shot line ascents with demand valve in hand already practiced by some instructors. This will significantly reduce, though not eliminate, the risks. As the vast majority of emergency situations are potentially avoidable, by watching the remaining air level and rigorously investigating all cases of equipment malfunction, great effort should be put into the reporting of all such incidents as may occur in order that dangers may be recognised and remedied before fatalities occur.

CONCLUSIONS

- 1. Scuba diving is a remarkably safe procedure, in part through the tolerance of the body to the majority of barotrauma incidents.
- 2. Cerebral Air Embolism occurs in a significant number of cases of carefully supervised SETT and Chamber pressure exposures but is rarely fatal. Minor cases are probably undiagnosed, but frequent.
- 3. Prevention of diving morbidity and mortality should be based both on a reduction of the likelihood of emergency situations developing and a thorough indoctrination of a course of action for any out-of-air situation. Such training should be less dangerous than the risk of the problem itself. Input of information is required to monitor both the causes of problems and the response/ outcome when they do occur.
- 4. 100% safety is never attainable. If practice in ascent is considered necessary, a shotline ascent with mouthpiece in hand seems the safest.

			TABL	<u>E 1</u>			
	AUSTF	RALIAN	"EMERGENCY	ASCENT"	PROJECT	1975	
PROBLEM	ASCENT	MODE:	Free Ascent	Buddy	Breathing	ī	TOTAL
Out of Air			17		9		26
Regulator failure		б		4		10	
Reserve failure			3		1		4
Hose supply failure		9		Nil		9	
"Mechanical"	Eailure		4		1		5
Sudden XS buoy	yancy		3		Nil		3
Other causes			5		Nil		5
Total			47		15		64

TABLE 2

EXTRA ALVEOLAR AIR AND AIR EMBOLISM ASSOCIATED WITH SETT TRAINING

New London (USA) and HMS Dolphin (UK) tanks

		SEA	FA	Buoyant	Steinke	Fatal	RCC	
							cases	
Moses	1928-51	1:16,100	1:1,030	1:3,250				
Peirano et al	1929-54	1:21,776	1:1,483	-	-	4	14	
Waite et al	1938-65	1:27,571	1:1,172	-	4	4		
Lambeth	1954-57	-	-	1:3,000	-	-		
Elliott et al	"20 yrs"		"better	than" 1:2	,300 asc	ents	5	3

Appendix A Reported Emergency Ascents

Report	Depth fsw	No Air Air	Reg Fail	Reserve Fail	Hose Supply	Other	Additional Details Supplied
A1	100 100	BB BB				FA	Mt Gambier: Octopus share ascent blackout near to surface accidental drop weight belt while teaching. Rough sea, poor visibility:- chest pain
A2 A3	80 ?	FA				FA	Tank entangled: murky
A4	20-40 20-40 20-40				FA FA FA		water at power station
	100 20	BB	BB				Mt Gambier: Subcutaneous emphysema resulted
A6 A9	? 60				FA	FA	"Mechanical failure of equipment"
A10	90 40 50	FA				FA	ditto
A11	100 ? ?			FA	FA FA		Mt Gambier Mechanical failure Mouthpiece dislodged
- 1 0	? ? ?	FA FA FA					
A12	? ? ?		FA FA	BB			
A13	120						All students use up air
A14	30				FA		Know of other hookah
A15	40					FA	Full facemask cracked, under "Leviathan"
	30	BB					Pupil, sea, low air; BB with Instructor
A16	60	FA					Freeing anchor after a dive
	20	FA					Reserve already "on" in error
	100	FA					Mt Gambier-recovering
	25					FA	"Some Idiot turned the air off"
A18	20		BB				occurred at a decompression stop
	150		BB				"dive required decompression so BB not FA"

Report	Depth fsw	No Air Air	Reg Fail	Reserve Fail	Hose Supply	Other	Additional Details Supplied
A19	30					FΔ	failed to fully open
>	00						on/off valve before
							water entry: know of
							other cases
	100			FA			Air cut off when pulled
							reserve: know of other
							cases
A20	120					FA	Interrupted air supply
	70	FA					Mt Gambier 2nd descent
							same tank; pulled
							reserve then no air.
							Untrained in FA.
A23	.70	FA					Gear exchange test:
	2	55					given empty tank
	?	BB					Horizontal shallow swim
7.04	120					ПЪ	till sale to ascend
AZ4	130					FA	Mc Gambier · ABLU Valve
	2					Π	Mt Cambier: ditto
⊅ 26	: 60				۳Δ	ΓA	Me Gambiel: Gitto
AZ0	150		BB		IA		Husband/wife team:
	190						wife's regulator failed
	100				* * FA		Hookah filter "blew-off
							screw" came free with
	100				**FA		two divers below. Both
							successful
	60				FA		Hookah hose changed to
							wrong outlet
	(100 s	norkelle	rs FA a	after divi	ng to sc	uba dive	rs at Mt Gambier)
В1	130		FA				Mt Gambier
- 4	60	FA					
B1	150	FA					
	150	FA					Shallow water black-
50	4.0						out": checked at SUM
BZ	40 25	FA	ΕN				
	25 150	ሮአ	ΓA				
	130	ΓA	ΨΔ				
B3	2	ፑፚ	ΓA				Night dive out of air
00	•	IA					after 5 minutes; with
							an instructor. "Often
							needed to FA since
							then: ALL divers do"
В4	?	BB					during a training
							session
	?					BB	faulty J-valve
В5	20	FA					"1-3 students run out
							of air every course"
	?	BB					"Student took my demand
							valve so BB with the
							instructor"

Some comments offered concerning practice of Emergency Ascents:

A2 possibly <u>allow</u> for 3rd class certificate, and <u>encourage</u> for 2nd class certificate A5 perform up shot line, demand valve mouthpiece in hand: RCC at site A6 with demand valve in hand and in all equipment, all present students. A10 7 foot ascent after ditch scuba, never in open water; allow, instructor present.

- All Allow, not obligatory, 6 metre depth water.
- A13 Require FA with instructor present; believe practically every diver on a shallow dive (20 feet or less) sometimes sucks tank empty knowing can FA.
- A14 Require ascent wearing equipment, mouthpiece in hand, from 60 feet. Avoid BB ascents. Would suggest ditching scuba or hookah if "for real" emergency
- A15 Desire that pupils practice but forbid because of legal liability risk.
- A16 Undecided: D&R in pool and sea but forbid solo FA. Suggest modify test to ditchswim to buddy and BB - return to and don scuba again, at constant depth
- A17 Essential but not desirable to inflict on students at initial training, include later. At least one FA from over 10 metres.
- A18 Teach pupils feel of an empty tank; forbid FA; teach NAUI bail-out not D&R
- A19 Require 3 metre D&R instructor monitored, pool and open sea.
- A20 Instructor demonstrates FA, prohibit pupils: horizontal swims set to set in 1 metre water suggested as good substitute for FA practice.
- A21 Instructor may show FA in shallow water: possibly ditch and ascend for more experienced divers, forbid for trainees.
- A22 Suggest increasing depths FA for 2nd class divers; forbid at basic level.
- A23 Instructor present allow ascent with mouthpiece in hand, then after ditch
- A24 Encourage FA in pool then sea with shot line, mouthpiece in hand
- A26 Forbid for pupils; suggest instructors train ascents 100 feet to 30 foot level. A27 Forbid beginners, possibly allow for 2nd class certification.
- в1
- Require all pupils to FA; never dive deeper than able to EFSA; 25 feet basic.
- В2 Require, even if only Ditch & Recovery test ascent: 15-20 feet.
- Require EFSA practice; criminally negligent if did otherwise: 10-40 feet В3
- Require but never more than 20 feet: would prefer 45-60 feet for basic divers В4
- Require, 10-15 feet increasing to 60 feet for advanced trainees. В5

Regarding practice of Emergency Ascent by all trainee divers:

A1, A2, A3, A4, A5, A6, A10, A11, A12, A13, A14, A15, A16, A17, A19, In favour: A22, A23, A24 B1, B2, B3, B4, B5.

Against: A7, A8, A9, A18, A20, A21, A25, A26, A27.

continued from page 15

	Spinal "bend" (air embolism?) with imperfect resolut	ion.	
Los Angeles	Cases include successful FA by minisub non-diver from	250 fee	≥t!
Nemiroff	Diver, entangled at 15 feet, dragged to surface by ten	der.	
	Diver, age 14, first dive, "deep", suddenly surfaced		
	Screamed & sank: rescued: decerebrate. Treated as		
	drowning, no RCC. No cure		
Okalyi	3 pearl divers FA from 15-30 feet	FA	TAL
Pollock	Holding onto fixed buoy; large waves washing over	FATAL	AE
USN 1972	5 feet training dive with semi-closed circuit scuba gi	ving	
	positive pressure gas supply. Probable AE signs.	CU	RED
USN 1971	35 feet depth using SSBN unit; free flowing regulator		
	while swimming. Snorkel diver 30 foot dive		
Uni Rhode Isla	and Numerous PBT cases are briefly reported in Scuba Safet	У	
	Report No 3		
Walker	Case SC 71/3 (unpublished) 6ft ascent in calm tepid wa	ter FA	TAL

APPENDIX B

SETT and Chamber cases of extra alveolar air and/or air embolism are noted in varying degrees of detail by a number of writers. Moses gives brief details of 71 incidents, of which 54 were with ascents and 17 in the pre-ascent pressure exposure test in a chamber at "112ft". There were in addition three cases of decompression sickness in instructors acting as attendants during therapeutic recompressions. This indicates a weakness in the therapeutic tables. Some of these cases are reported in greater detail by Behnke, Brown, Chrisman, Kinsey, Lieblow, MacClatchie, Polak and Adams, Polak and Tibbals, and Periano et al. A more recent case has been fully documented by Collins. In some of these US Navy cases some divergence from correct ascent procedure was either observed or later admitted but in others the drill appears to have been correctly performed. At all relevant times the trainees were under very close observation by highly trained and motivated instructors.

In the UK, SETT cases have been described by Elliott, Forbes, Honor, Lambert and Warner. Brief quotations may explain the strong views held by many Naval personnel involved in SETT training of submariners when advising against practice of out-of-air ascents by civil diving groups:

- "Accidents have happened to the actual training staff". (Warner, 1967)
- "The RN has considerable experience of free ascent training and reports that, in spite of the closest possible supervision, an appreciable number of incidents occur. Some of them, unfortunately, fatal". (Warner, 1969)
- "Case 1, a submariner, was a good swimmer in his early 20s. He volunteered to make a 100 foot ascent with instructors present. Wearing goggles, nose-clip and stole (life jacket) he made a "copybook" ascent. A loud exhalation of air was heard as he broke surface. He was unable to understand the order to put the tube back into the loop of the stole, caught the ladder with his left hand only, said "I feel..." and collapsed. It was only 6 feet to the RCC and he was compressed to "165 feet" less than a minute after leaving the tank bottom. He was conscious there so decompression was commenced. At 10 feet he said "When will I be able to see?" and it was realised that he was blind. He died 27 hours later". (Honor, 1970)
- "Despite all precautions, incidents occur. Over 20 years these have averaged 1 in every 2,500 ascents and have, in many cases, followed ascents which appeared to be in every respect normal and correct". (Forbes, 1975)
- "Since the adoption of free ascent with buoyancy by the RN in 1954 about 34,000 escapes have been made (15, 30, 30, 60 and 100 feet). There have been 10 casualties with symptoms primarily of cerebral air embolism and two with wide spread damage in the thorax without associated neurological symptoms, an incidence of only slightly more than 1 in 3,000 ascents. (Lambert ,1958)
- "The main group (studied) consisted of 112 subjects in which 4 cases of proven lung rupture and air embolism were observed. In addition to routine clinical investigations, EEG records were carried out before and after diving in the main series. It was found that free escape as such affects the EEG only slightly ... in some subjects without neurological symptoms (the changes) were so marked that the records following the diving were classified as abnormal." (Ingvar, Adolfson and Lindemark, 1973)

Caisson workers are apparently at risk of air embolism during their routine decompression after working under pressure, though the length of pressure exposure is not a critical factor. Warner reports that 6 cases occurred during the construction of the Dartford tunnel. They had been at less than 3 ATA for less than 4 hours. One victim who fell unconscious after leaving the lock, was later shown to have a lung

cyst. It was supposed that bronchial blocking was occurring in victims and tests were carried out on guinea-pigs that showed air embolism was produced in 75% of animals if bronchiolar obstruction was induced by a histamine spray in the chamber. Later experiments have shown air embolism can be produced in guinea-pigs after short exposure to 2 ATA. A case is noted where a fatal air embolism on decompression followed exposure to irritant fumes.

The German civil chamber incident was an example of the tragic results one can produce when dealing with forces one does not understand.

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APPENDIX C

Air Embolism cases associated with training

Cooperman	Pool - 9 feet ditch and recovery test	FATAL
Davis, Bassett	Lake - 35 feet swimming ascent training: unconscious.	
	Recovered "and 2 additional cases have occurred"	
Denney, Read	Pool - 15 feet lesson in buddy breathing failed	FATAL
Harveyson et al	Sea - 20 feet FA test. Bubbles in coronary arteries	FATAL
Hattori	Sea - 25 feet 3 cases during FA training.	
Kruse	"tank"? - ditch and recovery test. Hemiplegia but	
	recovered.	
Miles S	Sea - 20, 34, 35, 60 ft - Free Ascent training. 4 H	Fatalities
	Sea ? - 29, 25, 30 ft - FA training non-fatal	incidents
Nemiroff	Pool - 8 feet ditch and recovery test, at night.	Recovered
	Quarry ? - Free Ascent test: became unconscious	Recovered
Strauss,Prockof	Pool - 12 ft ascent followed by blindness, emphysema	Recovered
USN 12-70	Sea – 15 feet Buoyant ascent	
Uni Rhode Island	Pool	
(report 3)	- 8 feet Doff & Don test: breath help as ascent:blind	Case 1
	- 40 feet Doff & Don and FA	Case 4
	- 43 ft FA exercise: convulsed, paraplegic: recovered	Case 5
	- 90 ft FA exercise: part blind, right paralysis:	
	slow recovery	Case 15

Air Embolism diving incidents

Anderson	Helmet diver; rapid ascent from 30 feet FATAL
Davis, Bassett	Panic ascent (untrained) from 80 feet. Legs paralysed:recovered
	"and know of case from 10 feet"
Denney, Glas	Sea - 20 feet ascent. FATAL
	"19 such deaths in Michigan since 1959"
Elliott DH	Impaired consciousness after rapid ascent from 10 feet noted
Harpur	Sea - unconscious diver: buddy suffered fatal AE in rescue
	Sea - unconscious diver: bystander ascended, holding breath
	while buddy successfully rescued victim. Bystander got FATAL AE
Hattori	Sea - at 75 feet ascended 25 feet over a rock and suffered
	hemiplegia and disorientated. Rescued by instructor. Recovered
	fully. Also cases from 60 feet (fatal), 30 feet and 90 feet dives.
Jones	SA Navy diver ship bottom-search, on back; developed chest pain.
	Other cases PBT also reported
LaCarda	Betty G cleared mask forcefully while holding anchor line at 10 feet.

continued on page 13

Barotrauma after apparently normal diving

Bayliss G	Four non-fatal barotrauma cases. (Unpublished the	esis on
	underwater diving 1971).	
Colebatch	See previous reference	
Jones AG	See previous reference	
US Navy	Naval safety Report OPNAVINST 9940.2A 1971	

Cases of PBT associated with training

Cooperman et al.	Mechanisms of death in shallow water scuba diving. Canadian Med Assoc J 1968; 99(23): 1128-1133.
Davis, Bassett.	Diving Casualties, Lessons learned. NAUI Conference "IQ6", 1974
DDenney, Read.	Scuba Diving deaths in Michigan. JAMA 1965; 192(3): 220-222.
Harveyson et al.	Fatal air embolism from use of a compressed air diving unit. Med J Australia 1956; 21 April: 658-659
Hattori.	A review of air embolism among divers in the Monteroy Peninsular. SPUMS Journal 1975; July-Dec.
Kruse.	Air Embolism and other skin diving problems. Northwest Medicine 1963; 62: 525-527.
Miles S.	165 Diving Accidents. <i>J Royal Naval Medical Service</i> 1964; 50(3): 129-139.
Nemiroff.	The changing face of air embolism. NAUI Conference "IQ6", 1974
Strauss, Prockop.	Decompression Sickness among scuba divers. (Summitt & Berghage) JAMA 1973; 223(6): 637-640.
US Navy.	Research Report, 12-70
Walker D.	Unpublished report to "Stickybeak Investigation" (PNG Case)

Some diving incidents causing PBT, Air Embolism

Anderson WM.	Caisson disease during helmet diving. US Naval Med Bulletin 1927; 26(3): 628-630.
Denney, Glas.	Experimental studies in barotrauma. <i>J of Trauma</i> 1964; 4: 791-795,1964
Elliott DH.	Decompression, a hazard of underwater sports. J Roy Coll Gen Practit. 1969; 18: 233-237.
Harpur.	90 seconds deep scuba rescue. SPUMS Journal 1975; Jan-Mar <u>(also</u> NAUI News Jan 1974)
Hattori.	see previous reference
LaCerda.	Embolism disaster averted (case of Betty Gerzanics). Skin Diver magazine 1972; March.
Nemiroff.	See previous reference
Okalyi.	Occupational mortality and morbidity of divers in the Torres Straits. <i>Med J Australia</i> 1969; June: 1239-1242
Pollock.	A classic case of diver air embolism at the surface due to wave action. SPUMS J 1976; Oct-Dec (also PRESSURE April 1976)
Rose, Jarczyk.	Spontaneous Pneumoperitoneum after scuba diving. <i>JAMA</i> 1978; 239(3): 223.
US Navy.	Naval Safety Report OPNAVINST 9940.2A ref 1971 and 1972
Walker D.	Stickybeak Investigation of Australian Diving Deaths: some cases published in <i>Provisional Reports</i> , some await publication

Additional Sources

Los Angeles County. Underwater Safety Committee Report BS-AC. Diving Officers Conference Reports 1966-1977 (yearly) University of Rhode Island Reports

BIBLIOGRAPHY

Cases associated with SETT

 Behnke AR. Analysis of accidents occuring in training with the submarine "lung". USN Med Bulletin 1931; 30: 177-185. Brown EW. Shock due to excessive distension of lungs during training with escape apparatus. USN Med Bulletin 1931; 29: 366-370. Chrisman. Submarine escape training. J Connecticut State Med Soc 1938, 2(9): 423-430. Collins JJ. Unusual case of air embolism precipitated by decompression. New England J of Med 1962; 226(12): 595-598. Elliott et al. Clinical and Radiological features in 88 cases of decompression Barotrama. (Paper read at San Diego, 1975) Forbes B. Free Ascent: the position stated. Paper read at BS-AC Diving Officers Conference, 1975. Honor. Paper read at BS-AC Diving Officers Conference, 1970. Ingvar et al. Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study. James RE. Extra Alveolar Air resulting from Submarine Escape Naval Submarine Escape. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1958; 51(10): 824-827. Lieblow et al. Intra pulmonary air trapping in submazine escape training casualties in individual submarine escape. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape training. US Need Bulletin 1931; 29(3): 30: 165-177 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Wairner J. Paper to BS-AC Diving Officers Conference, 1967 	Adams BH.	Observations on submarine "lung" training. USN Med Bulletin 1931; 29: 370-372.
Brown EW.Shock due to excessive distension of lungs during training with escape apparatus. USN Med Bulletin 1931; 29: 366-370.Chrisman.Submarine escape training. J Connecticut State Med Soc 1938, 2(9): 423-430.Collins JJ.Unusual case of air embolism precipitated by decompression. New England J of Med 1962; 226(12): 595-598.Elliott et al.Clinical and Radiological features in 88 cases of decompression Barotrauma. (Paper read at San Diego, 1975)Forbes B.Free Ascent: the position stated. Paper read at BS-AC Diving Officers Conference, 1975Honor.Paper read at BS-AC Diving Officers Conference, 1970.Ingvar et al.Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study.James RE.Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968.Kinsey JL.Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255.Lambert RJ.Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827.Lieblow et al.Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289.MacClatchie.Medical aspects of submarine *lung" training. USN Med Bulletin 1931; 29(3): 357-366.Moses H.Casualties in individual submarine escape training. USN Med Bulletin 1932; 30: 165-177Polak, Adams.Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177Polak, Adams.Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865.Waite et al. <td>Behnke AR.</td> <td>Analysis of accidents occuring in training with the submarine "lung". USN Med Bulletin 1931; 30: 177-185.</td>	Behnke AR.	Analysis of accidents occuring in training with the submarine "lung". USN Med Bulletin 1931; 30: 177-185.
Chrisman.Submarine escape training. J Connecticut State Med Soc 1938, 2(9): 423-430.Collins JJ.Unusual case of air embolism precipitated by decompression. New England J of Med 1962; 226(12): 595-598.Elliott et al.Clinical and Radiological features in 88 cases of decompression Barotrauma. (Paper read at San Diego, 1975)Forbes B.Free Ascent: the position stated. Paper read at BS-AC Diving Officers Conference, 1975Honor.Paper read at BS-AC Diving Officers Conference, 1970.Ingvar et al.Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study.James RE.Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968.Kinsey JL.Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255.Lambert RJ.Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827.Lieblow et al.Intra pulmonary air trapping in submarine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289.MacClatchie.Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366.Moses H.Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964Peirano et al.Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955Polak, Adams.Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177Polak, Tibbals.Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865.Waite et al.Dysbaric Cerebral	Brown EW.	Shock due to excessive distension of lungs during training with escape apparatus. USN Med Bulletin 1931; 29: 366-370.
Collins JJ.Unusual case of air embolism precipitated by decompression. New England J of Med 1962; 226(12): 595-598.Elliott et al.Clinical and Radiological features in 88 cases of decompression Barotrauma. (Paper read at San Diego, 1975)Forbes B.Free Ascent: the position stated. Paper read at BS-AC Diving Officers Conference, 1975Honor.Paper read at BS-AC Diving Officers Conference, 1970.Ingvar et al.Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study.James RE.Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968.Kinsey JL.Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255.Lambert RJ.Submarine Escape. Proc R Soc Med J 1958; 51(10): 824-827.Lieblow et al.Intra pulmonary air trapping in submarine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289.MacClatchie.Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366.Moses H.Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964Peirano et al.Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955Polak, Adams.Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177Polak, Tibbals.Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865.Waite et al.Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966Warner J.	Chrisman.	Submarine escape training. <i>J Connecticut State Med Soc</i> 1938, 2(9): 423-430.
 Elliott et al. Clinical and Radiological features in 88 cases of decompression Barotrauma. (Paper read at San Diego, 1975) Forbes B. Free Ascent: the position stated. Paper read at BS-AC Diving Officers Conference, 1975 Honor. Paper read at BS-AC Diving Officers Conference, 1970. Ingvar et al. Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study. James RE. Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968. Kinsey JL. Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255. Lambert RJ. Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827. Lieblow et al. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289. MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	Collins JJ.	Unusual case of air embolism precipitated by decompression. New England J of Med 1962; 226(12): 595-598.
Forbes B.Free Ascent: the position stated. Paper read at BS-AC Diving Officers Conference, 1975Honor.Paper read at BS-AC Diving Officers Conference, 1970.Ingvar et al.Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study.James RE.Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968.Kinsey JL.Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255.Lambert RJ.Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827.Lieblow et al.Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289.MacClatchie.Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366.Moses H.Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964Peirano et al.Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955Polak, Adams.Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177Polak, Tibbals.Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865.Waite et al.Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966Warner J.Paper to BS-AC Diving Officers Conference, 1967	Elliott et al.	Clinical and Radiological features in 88 cases of decompression Barotrauma. (Paper read at San Diego, 1975)
 Honor. Paper read at BS-AC Diving Officers Conference, 1970. Ingvar et al. Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study. James RE. Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968. Kinsey JL. Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255. Lambert RJ. Lueblow et al. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289. MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Fraumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Wysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. 	Forbes B.	Free Ascent: the position stated. Paper read at BS-AC Diving Officers Conference, 1975
 Ingvar et al. Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study. James RE. Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968. Kinsey JL. Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255. Lambert RJ. Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827. Lieblow et al. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289. MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	Honor.	Paper read at BS-AC Diving Officers Conference, 1970.
 James RE. Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968. Kinsey JL. Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255. Lambert RJ. Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827. Lieblow et al. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289. MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	Ingvar et al.	Cerebral Air Embolism during training of submarine personnel in Free Ascent: an Electroencephalographic study.
 Kinsey JL. Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255. Lambert RJ. Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827. Lieblow et al. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289. MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	James RE.	Extra Alveolar Air resulting from Submarine Escape Naval Submarine Medical Centre Report No 550, 1968.
 Lambert RJ. Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827. Lieblow et al. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289. MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Fraumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Wysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. 	Kinsey JL.	Air embolism as a result of submarine escape training. US Armed Forces Med J 1954; 5: 243-255.
 Lieblow et al. Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289. MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	Lambert RJ.	Submarine Escape. Proc R Soc Med 1958; 51(10): 824-827.
 MacClatchie. Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366. Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	Lieblow et al.	Intra pulmonary air trapping in submazine escape training casualties. US Armed Forces Med J 1959; 10(3): 265-289.
 Moses H. Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964 Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Wysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	MacClatchie.	Medical aspects of submarine "lung" training. USN Med Bulletin 1931; 29(3): 357-366.
Peirano et al. Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967	Moses H.	Casualties in individual submarine escape. USN Submarine Medical Centre Report No 438, 1964
 Polak, Adams. Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	Peirano et al.	Submarine escape training experience. Med Research Lab US Navy Report No 264, 1955
 Polak, Tibbals. Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865. Waite et al. Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966 Warner J. Paper to BS-AC Diving Officers Conference, 1967 	Polak, Adams.	Traumatic air embolism in submarine escape training. USN Med Bulletin 1932; 30: 165-177
Waite et al.Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966Warner J.Paper to BS-AC Diving Officers Conference, 1967	Polak, Tibbals.	Fatal case of Caisson Disease following a dive of short duration to a depth of 30 feet. USN Med Bulletin 1930; 28: 862-865.
Warner J. Paper to BS-AC Diving Officers Conference, 1967	Waite et al.	Dysbaric Cerebral Air Embolism. Proceedings of 3rd Symposium on Underwater Physiology, 1966
	Warner J.	Paper to BS-AC Diving Officers Conference, 1967

General papers relating to pulmonary barotrauma

Colebatch et al.	Increased elastic recoil as a determinant of pulmonary barotrauma. Resp Physiol 1976; 26: 55-64.
Davies W.	Emergency Ascent for the Amateur. DIVE Underwater magazine December 1963
Griffin RJ.	A diagnostic sign of spontaneous interstitial emphysema of the mediastinum: case reports. Annals of Internal Med 1942; 17: 295-297.
Malhotea, Wright.	Arterial air embolism during decompression underwater and its prevention. <i>Proc Physiol Fen</i> 1960: 32-33
Walder D.	Some dangers of a Hyperbaric environment. Proceedings of 2nd International Congress Hyperbaric Oxygenation



DIVING MEDICAL SEMINAR Heron Island 23-30 September 1978

This will be the final Diving Medical Course to be held on the Great Barrier Reef. The format has been altered from the conventional style lecture courses of previous Queensland meetings. It will be based on the seminar/discussion style, held after the diving excursions, and following a brief factural revision by either Dr Robert Thomas or Dr Carl Edmonds.

Day 1	Registration -	Introductory Lectures by	Carl Edmonds Peter Tibbs Walt Deas
Day 2	Subject:	<u>Marine Animal Injuries</u> - Sharks & shark attack - Fish poisonings	
Day 3	Subject:	Common Diving Medical Disea - Barotraumas - Infections - Drownings - Photographic spot diagnos	ases sis
Day 4	Subject:	<u>Hyperbaric Medicine</u> - History - Current indications - Future trends	
Day 5	Subject:	Decompression Sickness - Pathophysiology - Treatment - Prevention	
Day б	Subject:	Medical Standards for Divir Hazardous diving areas	<u>19</u>

Case reports from both lecturers and other medical practitioners are very welcome.

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PULL THE OTHER LEG(S)?

Two patrons of a city hotel were startled by a large octopus on the floor of a ladies' toilet. The women told the management of the Hyatt-Regency Hotel they found the octopus outside a bar on the 22nd floor. The assistant manager, Mr Obie Collins, said the octopus was apparently placed there by two women dressed in army fatigues who were seen carrying a rubbish bag into the toilet. No one was hurt.

Australian, 24 April 1978

Did they check it was a female octopus?

A FUNDAMENTAL APPROACH TO THE PREVENTION OF DECOMPRESSION SICKNESS

SUMMARY

This article presents a hard look at the fundamental issues underlying the formulation of preventive decompression. The author's interest in this subject was captivated some fifteen years ago when taking instrument recordings of the remarkable decompressions routinely followed by pearl divers - particularly the Okinawans operating in the Torres Strait and elsewhere along the northern coast of Australia. This study revealed a very efficient decompression practice derived purely by trial and error at the expense of maybe several thousand lives and serious injuries. These remarkable practices were derived over half a century when that area supplied the world with the pearl shell which was in great demand before buttons were made of plastic. Working at Adelaide University the author and his aeromedical colleagues were just in time to put on record these practices before the pearling industry dwindled to a state at which that vast wealth of invaluable human experience would have been lost for ever.

The methods employed by those divers were both successful and much more economical on time than Naval practice. Moreover their emphasis upon spending much more time deeper at the start of decompression and surfacing directly from 25-35 feet was totally incompatible with the Haldane rationale and neo-Haldanian calculation methods for diving table formulation at the peak of popularity at that time. This discovery stimulated much scientific work at Adelaide, leading to concept of an equilibrium state rather than a supersaturated state as the most relevant in determining the imminence of bends. Publication of this approach in 1966 presented the first comprehensive challenge to the Haldane method of formulating decompression tables as elaborated by the US Navy in particular. The major point of divergence was to point out that only equations were used to formulate tables and that, whatever the accompanying words, conventional equations assumed that the bends-free dive was bubble-free, pointing out why the diver was so disadvantaged if this proved to be incorrect.

There is now much more scientific evidence to support the Thermodynamic Approach which has been updated recently in a book entitled "Decompression Sickness: The Biophysical Basis of Prevention and Treatment" (published by John Wiley's in New York and London). This paper is a distillate of that work. In order to avoid distraction from the main theme, some statements are made with minimal supporting data, if any, but the relevant references and detailed explanation can all be found in the book.

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The major medical problem in deep-sea diving is the prevention of decompression sickness, since any diver must decompress in returning to his normal environment and inadequate decompression can prove fatal or may lead to permanent disablement. By far the most effective way to avoid decompression sickness is to invoke gradual decompression; but this immediately raises the question of how gradual is gradual? Obviously one wishes to minimise the wearisome time spent by the diver in a chamber or suspended in the ocean and yet not jeopardise his safety. Consequently, a means is needed to optimise the whole environmental program needed to return the diver to the surface, ie. a simultaneous optimisation of:

- (DEPTH versus
- (TIME versus
- (COMPOSITION OF BREATHING MIX

The methods of accomplishing this fall into four broad groups:

- 1. Devise a schedule by trial and error.
- 2. Compute a schedule from a calculation method or mathematical model.
- 3. Use a meter based upon one of those models or calculation methods in #2.
- 4. Monitor a body parameter, using the response to determine decompression.

The last would be the best if a good parameter were available and the relevant tissue to monitor could identified anatomically. Although some encouraging advances have been made in monitoring tissues ultrasonically and conductometrically, such techniques must still be regarded as novelties until we can be sure of what to look for and where to look - issues discussed later.

The third approach (viz. the use of meters) is very good if the engineering is adequate but is really no more than a convenient form of #2 by providing a decompression unique to each particular dive history and so circumventing the fact that it is impracticable to compile a book of tables to cover all depth-time combinations. Most tables in operation today are hybrids of #1 and #2.

However, before discussing the formation of tables, a moment should be spent in considering whether it is worthwhile to calculate at all. Hence let us consider a major piece of purely experimental data - the bounce dive curves for air and heliox otherwise known as the no-stop decompression limits. Both depict a fundamental relationship between depth and time; so the fact that these curves can be so clearly defined for each individual can be taken as a manifestation of an underlying rationale which justifies efforts to them mathematically and even to invoke complex functions if needed. Behnke emphasizes this clear demarcation by saying that maybe 5 feet in depth can separate serious injury from a state of wellbeing.

In formulating a preventive decompression by means other than pure trial-anderror there are basically two approaches involving either:

- calculation methods in which a convenient equation is selected and constants determined empirically to offer the best fit to experimental data, adding more equations (and more constants) if needed, or,
- 2. true models based on the physiological and physical principles involved.

While the empirical approach (#1) is good for interpolating between dives already proven in the field, it has seldom been successful in extrapolating to greater depths or longer times. Thus a calculation method in which the constants have been adjusted to provide a safe table at 400 feet may fail completely when used to 500 feet. These discrepancies can always be accommodated by adding more hypothetical tissues (and more constants) until, with some 700-800 degrees of freedom which some designers use, one wonders whether it is worth invoking calculation at all. The calculation methods arising from the Haldane rationale have divagated into incredulous complexity to force a "fit" to experimental data.

The alternative approach - that of synthesizing a mathematical model from fundamental physics and physiology - would seem ideal until we realise how little we really know of the mechanism of decompression sickness. The symptoms are so varied that they tell us little; while pathological studies seem to show bubbles in most organs so, as Haymaker points out, nothing really pertinent to a specific model emerges from that vast mass of material.

However the symptoms do seem to fall into five broad categories:

- 1. Limb bends and other essentially *local* manifestations.
- 2. Cerebral symptoms which are rare.
- 3. Spinal "hits".
- 4. Vestibular DCS
- 5. Dysbaric osteonecrosis?

Dysbaric osteonecrosis

This disease induced by diving is little understood and, at this time, cannot be used to program decompression. Its principal features are:

- 1. No correlation between the incidence of bends and bone lesions when the data are analysed very carefully.
- No bone lesions in aviators at least, no more than the incidence in the "normal" population.
- 3. Greatly increased decompression time (eg. the Blackpool tables) greatly reduced the bends rate, yet did not change the incidence of bone lesions.
- 4. The time course for dysbaric osteonecrosis is several orders longer than anticipated for an acute insult at the time of the last dive.

As many as nine hypotheses for the mechanism of dysbaric osteonecrosis can be found in the literature, six based on acute infarction or vessel occlusion and three on a more subtle form of insult occurring at a more microscopic level of bone physiology.

In fact, it is just possible that dysbaric osteonecrosis may not be caused by the decompression

CNS Symptoms

Cerebral symptom are virtually identical to those caused by undisputed arterial air embolism, eg. when occurring after submarine escape, that their aetiology is seldom questioned. This is not true of spinal decompression sickness which occurs roughly three times more frequently. These CNS symptoms can always be produced by a decompression far in excess of one known to induce mild limb bends, yet it is probably fair to say that they are rarely the presenting symptoms for *marginally* unsafe decompressions The factors predisposing the subject to the rare exceptions to this general rule are discussed later (p. 25).

Limb Bends

Most empirical calculation methods work to a "trigger point" for each hypothetical tissue, eg. violating an 'M' value in the conventional approaches. Calculation effectively stops at that point as though whatever is "triggered" must occur. However, let us consider the man performing a dive on which he has developed bends some time after return to the surface. He now repeats that exposure, presumably violating the same hypothetical "trigger points", but recompresses to 20 feet shortly before he knows he will develop bends (and *limb* bends are quite reproducible*), stays there for 30 mins and then returns to the surface with no problem. The recompression to 20 feet for 30 mins obviously averted what would otherwise occurred, so the process leading to bends must have taken place in at least two steps:

^{*} generally occur in the same individual for the same exposure and decompression.

(1. a primary event "triggered" by decompression

(2. a critical insult producing symptoms

(

Moreover, there would appear to be a continuous variation in the insult, bends occurring only if it reaches a threshold level for pain. See Figure 1.



FIG. 1

This raises questions concerned not only with identifying these processes but with other queries needed to be answered in formulating a mathematical model from fundamental considerations. This list of questions includes:

- 1. What is the primary event?
- 2. What is the mode of insult and what is its critical level for pain?
- 3. What conditions initiate the primary event?
- 4. What is the cause of delay in reaching the critical

insult?

- 5. How is gas taken up by tissue?
- 6. How many tissues are involved?
- 7. Does the prevention of limb bends avoid other forms of decompression sickness and what factors tend to predispose the diver towards those symptoms?





Primary event

Let us consider a simple exposure to a pressure P_1 followed by a decompression to a pressure P_2 - see Figure 2. Now the likelihood that limb bends will occur at P_2 is determined by numerous factors which can be reduced to two primary ingredients:

1. The extent of the decompression $(P1-P_2)$

- 2. The inert gas content immediately prior to decompression as determined by:
 - (a) time "on the bottom",
 - (b) depth of exposure,
 - (c) solubility of inert gas breathed (eg. nitrogen or helium),
 - (d) exercising "on the bottom",
 - (e) substituting oxygen for inert gas in the breathing mix, etc.
 - (f) obesity (increased body fat)

The dominance of these two factors leaves few alternatives, if any, to the popular view that the primary event is the inception of a stable gas phase.^{*} It is very difficult to conceive other initiating processes which are so dependent upon the *combination* of these two dominant features listed above without invoking the principle of gas separating from solution. So far this introduces no controversy since, ostensibly, all designers of calculation methods and models claim that their indices for limiting decompression are thresholds for bubble inception; although whether they do so in practice is quite another matter. However this agreement ends abruptly when we proceed to the next question (#2 on p. 4) and consider the mode of insult leading to limb bends.

Mode of insult

Several mechanisms have been proposed or assumed by which the primary event can lead to the critical level of insult needed to induce limb bends. These differ according to the type of pain - whether induced by ischaemia or by mechanical means - and whether the insulting entity is a bubble or one of several degradation products known to be produced by a gas-blood interface. These approaches can be summarised as follows:

ischaemia		intrav	vascular	bubb	oles
	by		OR		
OR		blood	degradat	cion	products

PAIN INDUCED by

mechanical		extravascular g	as phase	Ē
	by	OR		
means		tribonucleation	(joint	gas)

Of these, tribonucleation is most unlikely since gas injected between the articular surfaces of the joint, or formed by various disease processes, does not induce pain (aeroarthrosis). In limb bends the pain is not within the joint but around it. Moreover it requires foreign particles much harder than bubbles to penetrate the synoviun or articular surfaces to the depth of any nerve endings - such as sodium ureate crystals in gout and the pain of gout is much different to that of limb bends.

^{*} we are simply concerned with whether bubbles form or not and mechanism(s) of nucleation/activation of nuclei, etc. are of largely academic interest.

Returning to the other mechanisms, the pain of *limb bends* is unlikely ischaemic in origin for the following reasons:

1. Ischaemic pain must be induced by *arterial* emboli, but bubbles only form *de novo* in the *arterial* system with explosive decompression, while venous bubbles are trapped by the lungs.

2. Known arterial air embolism does not produce limb bends, eg. after an accident in submarine escape training.

3. Diseases known to produce infracting agents such as thrombi, fat emboli, platelet aggregates, etc. do not produce the pain of limb bends.

4. Compression affords immediate relief of pain in most cases of limb bends while ischaemic pain is greatest upon restoration of blood flow.

5. If pain were ischaemic in origin, one would expect that further decreasing tissue oxygen supply by lowering the inspired oxygen partial pressure would exacerbate the situation, and yet hypoxia has been found to have a mild protective action - if any.

6. Similarly one would expect elevated oxygen to help relieve ischaemic pain and yet hyperoxia *per se* (as opposed to oxygen substitution for inert gas) potentiates the bends.

This would lead us to deduce that the mechanism of limb bends is a bubble pressing on a nerve ending.

Mechanical approach

This simple approach to the pain of limb bends implies that the gas would probably need to be located in an extravascular site in order to bend a nerve ending as far as its pain threshold. This is easily justified by the fact that, even after an extensive recompression for a few minutes, limb bends will re-occur in the same site upon a further decompression same pressure at which they occurred in the first place. Intra-arterial bubbles actually observed invascular window preparations can be totally displaced by such recompressions and washed away in the circulation, gas causing the local pain of *limb bends* is fairly certain to be extravascular.

In selecting an extravascular site for the insult, it becomes more important to identify a tissue anatomically since it would then eliminate major problem of programming a decompression by following a direct tissue monitor - viz. the question of knowing where to look. It would also provide for the physiological parameters in the model. Thus the requirements the critical tissue type can be listed as follows:

- 1. a preponderance of nerve endings;
- insult of those nerve endings by other mechanical means must provoke a pain similar in nature to bends;
- there should be a positive correlation between gas content and bends in that tissue;
- 4. it needs to be a "tight" tissue since a compliant tissue would enable gas to expand freely when its pressure would be less likely to reach the pain threshold for bends (Figure 1);
- 5. a small change in blood flow with exercise of that limb.

All of these conditions have been satisfied by tendon, but could also apply to certain other connective tissues.

Pain threshold

The simple mechanical concept of pain (in limb bends only) has been particularly well demonstrated by Inman and Saunders who found that the identical pain could be produced by injecting Ringer's solution into tendon and other connective tissues. This pain was not determined by the volume of the solution injected but by the pressure differential. Moreover it was reversible and appeared or disappeared at the same pressure threshold

If we return to the situation of a gas rather than Ringer's solution pressing on that nerve ending, the net deforming pressure is the net gas pressure (internal less interfacial effects - δ_g) plus pressure (δ_f) due to any fluid accumulation. Thus bends pain can occur if:

$$\delta_g + \delta_f > \delta_t \qquad \dots \qquad (1)$$

This very simple criterion for pain can be related to the volume gas (v) separated from solution in unit volume of tissue by:

 $\delta_{g} = Kv \qquad \dots \qquad (2)$

where K is the modulus (reciprocal of compliance) resisting expansion of the tissue.

Thus the unknown $\delta_{\,q}$ can be eliminated, so that

BENDS can occur if:
$$v > (\delta_t - \delta_f)/K$$
 ... (3)

This simple quantitative relationship is compatible with the fact that limb bends are more likely to occur for a greater exposure or decompression $(\nu\uparrow)$, in an older subject $(K\uparrow$ with age) for nerve endings sensitized by release of serotonin or other humeral factors $(\delta_t \downarrow)$ and trauma $(\delta_f \uparrow)$ while ameliorated by acclimatization $(K\downarrow$ with creep) or plasma expanders $(\delta_f \downarrow)$. Moreover, in absolute terms, the δ_t value from Inman and Saunders combined the value of vestimated for a diver whose minimum bends depth is fsw gives a K value within 10% of that for excised tendon.

Relation to dive parameters

While the simple mechanical model can interpret many of the widely differing features of limb bends, we need to know whether this extends to the parameters of a dive.

Let us again consider the simple case of an exposure to a pressure (P_1) by immediate decompression to P_2.

If the nitrogen tension at P_1 is P_{N_2} immediately prior to decompression and is then reduced to P_{N_2} by 'dumping' nitrogen into the gas phase until a quasi phase equilibrium is established at P_2 , then a simple nitrogen balance gives:

n P _{N2} =	s _{N2} .P _{N2} -	$S_{N2} \cdot P_{N2}$	(4)
(N ₂ dumped	$(N_2 \text{ initially})$	$(N_2 left$	
from solution)	in solution)	in solution)	

where $\ensuremath{S_{\mathrm{N2}}}$ is the solubility of nitrogen in the tissue.

This now relates ν to the nitrogen tensions before and after decompression. Before decompression:

 $P_{N_{2}} = F_{IN_{2}}(P_{O}-P_{W}) + F_{IN_{2}}(P_{O}-P_{W}) \dots (5)$ (nitrogen before (nitrogen taken up compression to P_{1}) in time t at P_{1})

where P_0 is normal atmospheric pressure, P_W is water vapour pressure at body temperature, F_{IN2} is the volume fraction of nitrogen at body temperature and \emptyset^t is the function of time (t) for uptake [for the particular case where a steady state has been reached at P_1 , \emptyset (t) = 1]:

Steady state:
$$P_{N2} = F_{IN2}(P_1 - P_w)$$
 ... (6)

The only remaining unknown in relating the pain threshold δ_t to dive parameters is now P'N2. This can be resolved by a simple pressure balance – but only for the "worst possible case".

Worst Possible Case

This is the state where at least one out of many millions of micro-regions of the tissue has 'dumped' all gas in excess of saturation to come to phase equilibrium. This is the worst possible not only because it represents the maximum volume of gas which can separate from solution, but there is then the lowest driving force remaining to eliminate that gas from the tissue via blood - see p. 18.

In any bubble the absolute gas pressure is determined by the external pressure, the pressure needed to push tissue aside in forming the bubble and the effect of surface tension (γ) as described by the Laplace equation ($2\gamma/rSDO5(b)$). According the Dalton's Law this total absolute pressure must equal the sum of the partial pressures as depicted in Figure 3.

 $P'_{N2} = P + B - m$...(7)

where B is a small constant as defined in Figure 3 and $m = P_{\rm VO2} + P_{\rm VCO2} + P_{\rm W}$ and is approximately constant provided $P_{\rm VO2}$ does not exceed about 100 mm Hg.



Summation in Bubble

Figure 3

The sum of the mechanical contributions to bubble gas pressure must equal the sum of the partial pressures of all gases present - Dalton's Law

Decompression ratio

In the past much attention has been paid to the use of decompression ratios of `M' values, so it is important to see whether the simple mechanical approach to bends pain can explain the apparent adherence of decompression limits to a ratio concept.

Let us again consider the simple case (Figure 2) of a diver who has attained stead state at P_1 being decompressed to a pressure P_2 (ie. $P = P_2$ in equation 7). Elimination of unknowns in equations 3, 4, 6 and 7 gives the simple relationship:

 $P_{1} = WP_{2} + Y \dots (8)$ where $W = (v + S_{N2})/S_{N2} \cdot F_{N2}$ $Y = [(v + S_{N2}))(B-m) + P_{w} \cdot F_{IN2} \cdot S_{N2}]/S_{N2} \cdot F_{IN2}$

which is a small constant if $\boldsymbol{\nu}$ is constant.

Much more important is the fact that the gradient (W) is constant if the volume (ν) is constant.

Equation 8 can be re-written in terms of a ratio (M = PSDO5(1) / PSDO5(2)) as:

 $M = P_1/P_2 = W + Y/P_2 \dots (9)$

Thus the simple mechanical approach gives a linear relationship between P_1 and P_2 - in fact, almost a ratio since Y is small. It predicts a decreasing ratio ($M\downarrow$ as $P_2\uparrow$) as many proponents of popular calculation methods now prefer.

Actually a linear relationship offers a better separation of experimental bends and no-bends points as seen in Figure 4.

It also offers a much better correlation between diving and aerial bends where, by a simple ratio, a value of around 2 would apply for divers but 3 for aviators.

PULL THE OTHER LEG(S)?

Two patrons of a city hotel were startled by a large octopus on the floor of a ladies' toilet. The women told the management of the Hyatt-Regency Hotel they found the octopus outside a bar on the 22nd floor. The assistant manager, Mr Obie Collins, said the octopus was apparently placed there by two women dressed in army fatigues who were seen carrying a rubbish bag into the toilet. No one was hurt.

Australian, 24 April 1978

DID THEY CHECK IT WAS A FEMALE OCTOPUS?



Figure 4 Data from Hempleman (1957)

Number of tissues

Returning to the list of issues (p. 4) for which answers must be found or assumed in the development of any model based upon fundamentals, the next question concerns the number of tissues, or rather, tissue types involved in limb bends. This is important since it determines the number of independent constraints to be applied to the formulation of the decompression and, hence, the number of independent equations to be used.

One might expect that, if the Haldane rationale applied, then "triggering" the 10 min "tissue" would provoke a different response to "triggering" the 40 min "tissue" and yet no correlation between the symptom and any hypothetical "tissue" has ever been shown. Hence there would seem to be no good reason for assuming more than one tissue- at least, no more than one *anatomical* tissue is involved in *limb bends*.

If there were several tissues, then one would expect a transition point in the dose-time curve whenever one superseded another in the imminence of pain, ie. a 'kink' in the no-stop decompression limits and yet none is perceptible in the data for air or heliox.

Similarly, if we return to the case of simple decompression from P_1 to P_2 where P_2 is 'titrated' to marginal bends, then we would expect a transition if one tissue were to 'take over' from another as bends-determining. however, once again, no transition point could be detected until the pressure was in excess of about 300 feet (Figure 5) when the P_1 vs. P_2 relationship follows a different linear relationship, but vestibular DCS are then the presenting symptoms. Hence another tissue must be included for depth of over 300 feet, but there is then ample justification from the symptomatology. However, for *limb bends* there would still appear to be no reason to assume that more than one tissue type is involved - and *tendon* would seem a likely candidate according to previous discussion (p. 8). The next question concerns how this tissue takes up inert gas.



Figure 5

Data from Hempleman (1975).

How is gas taken up by tissue?

So far we have considered only those decompressions where the man has reached a steady-state before decompression, ie. where his pre-decompression nitrogen tension is independent of time as expressed by equation 6.

However, for shorter bottom times or further decompression to shallower depths, it is necessary to estimate tissue gas content in the light of the past history, ie. $P_{\rm N2}$ is now a function of time. The question is then which function do we use. It is obviously an asymptotic function to allow for attaining steady state, but there are thousands from which to choose and the popular exponential (as used in the Haldane rationale) is just one possibility.



Figure 6

Various models which have been used to try to quantify inert gas uptake.

To try and answer this question from fundamentals we need to determine the appropriate model (Figure 6) for gas exchange in the critical tissue and this immediately raises the very fundamental question concerning whether the uptake of an inert gas is limited by the circulation (blood perfusion rate) or by diffusion when the relevant diffusion barrier can be either a membrane or the whole bulk of cellular material - see Figure 6 for alternative models which have been proposed.

To put these in perspective, let us consider and individual who has just switched from air to heliox breathing at normal pressure. The macro distribution of helium will be effected by the circulation while its subsequent assimilation by the extravascular tissue must occur by diffusion. Thus blood perfusion and extravascular diffusion are two transport processes in series, but which is rate-controlling?

There is a vast literature relevant to this issue, but very little can be considered decisive. However, to put many of these studies in perspective, we might return to the above case of the individual who has just switched from air to heliox. The highest tension of helium will occur in arterial blood (p_a) while mean tissue $(-p_t)$ will be the lowest. One can then argue that if the tension is at the interface between the perfusion and diffusion transport processes is closer to arterial, then uptake is largely diffusion-limited; otherwise, if this tension is closer to mean tissue tension, it is largely Unfortunately this gas tension at the capillary wall perfusion-limited. cannot be measured, but venous (p_v) may be taken as a good reflection of it. Hence the fact that Kety and Schmidt found that venous tension (pa) lay so close to their estimated mean tissue for N_2O in monkey brains was taken as strong evidence that uptake was controlled predominantly by the circulation. This has provided the basis for the conventional concept that blood perfusion is the rate-limiting process and that venous blood leaves in equilibrium with tissue $(p_v = -p_t)$. This gives a simple exponential function as the time response for a single tissue as needed by the Haldane rationale for decompression formulation. The membrane model (b in Figure 6) would also predict an exponential.



Figure 7

Arterial (p_a) , venous (p_v) and mean tissue $(-p_t)$ tensions of inert gases in brain monitored after a sudden switch to that gas in the breathing mix.

However, more accurate analyses monitoring isotopes indicate that venous tension starts by following ACOIVS1(-,p)SDO9(t) but then moves close to pSDO5(a) with time (Figure 7), ie. uptake starts perfusion limited as the first of the new gas enters the system and then changes to predominantly diffusion limited as the increased assimilation reduces the gradients. However the same data can be equally well interpreted by saying that venous is the mixed venous outflow from many zones of the same tissue, so the only conclusion is that the final model is more than either (a) or (b) in Figure 6, and is either:

- heterogeneous perfusion of the critical tissue in which each micro-region would have its own half-time (but this would still not explain a different M value needed for each), or
- the rate-limited mode of uptake is bulk diffusion (model C in Figure 6), or
- 3. uptake is controlled by both processes.





Figure 8

If we consider the most uniformly perfused organ large enough to viz. isolated skeletal muscle, the classical studies of Renkin have that the uptake of inert tracers is circulation-limited at low blood perfusion rates and diffusion-limited for high flow rates. Unfortunately, the normal physiological range lies intermediate between these two limiting cases so that neither diffusion nor perfusion can be ignored. This probably applies to the critical tissue also, so a compromise between the two is envisaged - Figure 8. This is the Kety concept of the stirred tank but taken only as far as the walls of the cell - immersed as though it were an irregular shape of effectively uniform permeability in which gas is assimilated by relatively slow bulk diffusion.

Summary

So far, this discussion has been concerned with the mechanism for the occurrence decompression sickness rather than its prevention. This has indicated that the pain of limb bends has a simple mechanical basis which is easily quantified and most likely refers to just one anatomical tissue type (probably tendon or another tight well innervated connective tissue) in which gas uptake is controlled by both the blood perfusion rate and diffusion into the bulk of extravascular tissue. The next step is to see how this simple model can be used to optimise a decompression, but it was first necessary to test its compatibility for non-optimal situations. After all, you may disagree with the way the other fellow formulates his decompression, but you must still predict the outcome of the trials of his method by your model.

OPTIMISATION

Let us consider a diver who has just completed his task on the bottom wishes to return to the surface safely yet without wasting time unnecessarily. The immediate question is how far does he decompress on the first 'pull'? Does he decompress all the way to a level just deeper than his bends point or is there some other criterion which determines his optimal depth?

If that particular phase of the decompression would enable the man to surface then the answer is obviously 'yes'. However, if bends would occur before surfacing, then do we decompress almost to his bends point or stop much sooner?

Conventional 'supersaturation' approaches to decompression, as presented in the multitude of neo-Haldanian calculation methods, assume that no gas phase is formed if you do not exceed the "trigger points" as expressed by ratios or 'M' values. The all-important question is does a sub-symptomatic decompression initiate the gas phase? By the model developed in this discussion, we may have a good correlation between the incidence of bends and other factors for single decompressions, but when does the primary event actually occur?

Point of inception of gas phase

There is three basically different approaches to describing the point of inception of a stable gas phase in tissue:

- 1. The bends point
- 2. Point of phase equilibrium, and
- 3. Some intermediate "trigger point".

If the second of these is true, then it is most disturbing since it implies that tables formulated by the other approaches are provoking bubbles including most conventional diving tables and US Navy tables in particular. However, before attempting to pursue this matter, it is desirable to know why this is such an important issue. After all, why should a few "silent" bubbles be so serious if they are not causing pain or other symptoms?

Importance of supersaturation vs. equilibration

for nitrogen elimination from tissue, whether it is:

The intention of all approaches to decompression formulation is to select conditions which will give the maximum rate of elimination of inert gas from tissue at each moment. Comparatively little can be done to change the resistance to the transfer of the gas, but a great deal can be done to select the optimal driving force for transfer of the tissue inert gas to blood for its elimination via the circulation, eg. $\Delta PSDO5(N_2)$ for air diving.

Driving force for N_2 elimination

The gradient for nitrogen elimination is simply the tissue-blood gradient where the blood tension for an arbitrary absolute pressure P is given by F_{IN2} (p-p_W) as per equation 6. The real problem is the value to use for tissue N₂ tension, ie.

 $$P_{\rm N2}$$ for gas remaining dissolved, or whether $$P'_{\rm N2}$$ where gas in excess of equilibrium has been 'dumped'

This leads to two very different equations for the all-important driving force

IN SOLUTION
$$\Delta P_{N2} = P_{N2} - F_{IN2}(p-p_w)$$
 ...(10)

OR

WITH SEPARATION: $\Delta P_{N2} = P(1 - F_{IN2} + B - m' \dots (11))$ where $m' = m - p_W F_{IN2}$

This comparison is extremely important since further decompression (P \downarrow) would increase $\Delta P_{\rm N2}$ if all gas remains dissolved but decrease it if there is phase separation, since phase $F_{\rm IN2}$ must be less than 1 (0.8 for air). Thus the popular practice of decompressing as far as possible on the first 'pull' towards the surface, so characteristic of US Navy schedules, could have the diametrically opposite effect to that intended. In other words "by getting the hell out of it" on that first long pull towards the surface, the driving force for nitrogen elimination is actually being *decreased* rather than *increased* - IF phase equilibration best describes the primary event. It therefore becomes imperative to establish whether a sub-symptomatic decompression can cause gas to separate from solution in the critical tissue(s).

"Silent" bubbles

There is now overwhelming evidence that bubbles can be present during asymptomatic decompression. This includes examinations of sacrificed vascular window, X-ray studies, measurement of cerebrospinal fluid volume, conductometric monitoring of tissue and ultrasonic surveys. Even a device as crude as the ultrasonic bubble detector based on the Doppler principle indicates a host of venous bubbles in asymptomatic divers - often after the first long 'pull' towards the surface if using USN schedules.

However it can always be argued that these intravenous bubbles are irrelevant - particularly if we adopt the mechanical approach to bends pain discussed earlier. After all, nitrogen is five fold more soluble in fat and, from a structural standpoint, adipose tissue can be considered weak. Hence it is easy to envisage the large volume of extravascular gas bursting the capillary wall depositing bubbles, fat emboli and portions of endothelial cells into the circulation where they appear about the same time. Electron micrographs have been taken showing extravascular gas entering capillary blood in cutaneous tissue. The large volume of nitrogen would not give pain in adipose tissue due to the lack of nerve endings. Thus Doppler sounds probably refer to fatty tissues which would reflect the state of the critical tissue to some extent - hence the poor yet positive correlation between bends and 'venous' Doppler sounds.

It would therefore seen more relevant to turn to studies where a "tight" well innervated connective tissue has been monitored.

Conductometric studies

The electrical conductivity of rat tail has been monitored during decompression, this being predominantly tendon. Electrical resistance was found to increase, the magnitude of the increase being greater for those animals which had respired a more soluble inert gas. Whereas this and the reversal upon recompression left no doubt that the electrical changes were caused by the separation of gas from solution, the interesting feature was the absence of any change until a minimal decompression of 95-145 mm Hg had been reached - whatever the inert gas present (Figure 9).

This threshold decompression for the appearance of the gas phase happens to coincide with the position of phase equilibrium in extravascular tissue (Figure 10) and agrees well with the altitude for the onset of bubbles as seen by X-rays.



Figure 9 Electrical conductivity of a rat tail monitored during decompression to altitude at a uniform rate of pressure change. Data from Hills (1971).



Inherent unsaturation

This immediately raises the question of why the position of phase equilibrium differs from normal atmospheric pressure in a subject who has always breathed normobaric air and might be considered "saturated". The reason is that, while Dalton's Law of partial pressures must apply to the gaseous phase, it need not hold in liquids; so that the conversion of a relatively insoluble gas (0_2) into a much more soluble one (CO_2) by metabolism causes a permanent deficit in the total gas tension of tissue relative to absolute pressure. This inherent unsaturation is depicted by the shaded area in Figure 10. Such reasoning implies that the term "saturation" diving is a misnomer and "steady state" might be more appropriate, since only a dead diver could reach true saturation before the start of decompression. The inherent unsaturation is very important not only because it determines the position of phase equilibrium upon decompression, and hence the point at which the first bubbles can start to form, but it provides a permanent driving force for dissolving gas in the body. This includes not only bubbles but intrapleural gas, gas in an occluded bronchioles or a blocked sinus, etc.

It has been demonstrated very simply by a sealed subcutaneous tube made from a non-collapsible plastic permeable to all gases and water vapour. Over a period of a few hours, the tube develops a partial vacuum of 80-100 mm Hg and stays at that value ad infinitum. Moreover, if the breathing mix or pressure is changed it moves to another value determined largely by the magnitude of the inspired P_{O2} . Thus the subject who has breathed pure O_2 for a few hours at normal pressure has an inherent unsaturation of 600-700 mm Hg, so that he can decompress by this amount without any fear of inducing bubble formation.

Moreover the unsaturation also provides the driving force for dissolving bubbles. Let us consider the transcutaneous tube which has reached a steady state by virtue of all gases and water vapour equilibrating with the adjacent tissue. If the rigid tube were suddenly removed, the gas would be compressed by the inherent unsaturation and this compression would disturb the equilibrium and, in so doing, provide a driving force for dissolving the gas equal in magnitude to the original inherent unsaturation. Thus the inherent unsaturation is particularly desirable and fundamental to the whole problem of formulating decompression.

Decisive tests

The foregoing evidence indicating that gas could separate from solution in tissue for much lesser degrees of supersaturation, if any, than implied in conventional calculation methods led this writer to claim that such diving (including USN schedules) were not preventing bubbles but were really *treatment* tables for containing subsymptomatic gas below the pain-provoking threshold. However, such a serious implication needed a definitive test, since previous experiments to try to settle this issue had involved searching for gas in one way or another and all such d*irect* methods may have been monitoring an irrelevant tissue.

A crucial test which avoids identifying the critical tissue anatomically

has exploited the difference in $\Delta PSDO5(N_2)$ depending upon whether gas remains in solution (equation 10) or is 'dumped' into the gaseous phase (equation 11), the significance being emphasized on page 18. Let us therefore consider a man who has spent one hour at 160 feet on air and has followed the appropriate US Navy air table to the end of the time normally allotted to the 20-foot stop - Figure 11.

Figure 11

Titration of a last stop at 10, 20 or 30 fsw on the same goats. Data from Hills (1968).

If no bubbles have been formed to that point, as assured in the formulation of the table based upon $P_{\rm N2}$ rather than $P'_{\rm N2}$, ie. by neo-Haldanian reasoning, then decompression to the 10-foot stop at that time should provide more driving force for nitrogen elimination (equation 10) and hence a safer decompression. On the other hand, if much gas separated from solution on that first long 'pull' to 60 feet, and the subset decompression has simply been "treating" them, then we should use $P'_{\rm N2}$ and equation 11 rather than equation 10 to determine the true outcome. It would then be better to remain at 20 feet when the driving force is greater than at 10 feet $(\Delta P'_{\rm N2}^{\uparrow})$ as P[↑] in equation 11) and surfaced directly from 20 feet. When total decompression times were 'titrated' to bends

points on the same animals, it was found to be more efficient to surface directly from 20 feet than to include a 10-foot stop (see Figure 11) indicating that the gas phase *is* present *in the critical tissue* during this particular USN decompression and probably during many others based on the same calculation method. This finding is compatible with the observation common in aviation that preoxygenation is much less effective in eliminating nitrogen if undertaken at altitude as opposed to ground level (ie. $\Delta P'_{N2} \downarrow$ as P↓ when the gas phase *is* present).

This point has been emphasized since it points to a very fundamental and significant inadequacy in conventional calculation methods used to formulate diving tables, ie. any separation of gas from solution can greatly reduce its rate of elimination from tissue.

Optimisation

Well, it is easy to be critical, but does the concept of the 'worst possible case", ie. phase equilibrium and the inherent unsaturation, really offer any better alternative? This writer believes that there is a reservoir of nuclei in tissue with a spectrum of energies for their activation into stable bubbles capable of growth and, hence, the inception of bubbles is a somewhat random process. However, whereas most areas retain their supersaturation, it only requires one out of maybe many million micro regions to 'dump' its gas for limb bends to occur. Thus the "Thermodynamic" approach considers this 'worst possible" case to be the most relevant. This concept has subsequently been re-named "Nil Supersaturation", and "Zero Supersaturation" by other workers.

The criterion for optimisation is therefore one of avoiding any supersaturation and yet not wasting time in decompression, ie. keeping the one tissue for limb bends just on the point of true saturation. However, if bulk diffusion is one of the resistances to gas transfer, we must apply the phase equilibration rule to each point and not just to the value of total gas tension averaged over the whole tissue. Thus we must estimate the peak total gas tension and then decompress by reducing pressure until it coincides with this peak (Figure 12). Thus the decompression continues until the diver has the amount of gas in his tissue which he could tolerate on the surface. At this point (usually around 20-25 feet) he "drops out" and forms the gas phase but to just below the pain provoking volume as defined by equation 3.

Figure 12

HOW ANEMONEFISH SURVIVE SEA ANEMONE NEMATOCYSTS

Doug Wallin has reported (<u>Sea Frontiers</u>, 24(1), 1978) recent studies of this surprising survival of anemonefish in its chosen habitat. The mucus covering the skin of each fish contains an inhibitor chemical that prevents the nematocysts from discharging. The fish acquire this ability after birth, lacking this immunity when first settled from the plankton. This takes about an hour, during which time it repeatedly brushes briefly against the tentacles. The anemone tentacles themselves must obviously contain a similar chemical to avoid stinging each other into impotence.





The Thermodynamic (Equilibrium) concept of decompression formulation.

The net effect is to introduce much deeper stops and redistribute decompression time towards the deep stages by comparison with conventional neo-Haldanian approaches - see Figure 13. Thus the Thermodynamic approach pioneered the concept of deep stops which have subsequently been introduced into most commercial tables by trial and error. At least, it provided a theoretical basis to justify the empirical modifications found necessary in order to reduce the high bends rate which those calculation methods were otherwise incurring. It also explains the much more efficient methods devised over years of trial and error by Okinawan pearl divers operating off the Northern coast of Australia.

Other Symptoms

So far we have concentrated upon limb bends on the basis that their total avoidance also avoids 99% of other symptoms. However this is not entirely true, so we now need to look at the predisposing factors and then try to avoid them within the framework already outlined for programming the decompression based on limb bends.



Figure 13

We cannot be sure of all the predisposing factors but two which seem to be emerging are:

- Avoid arterial bubbles which can lead to cerebral and, possibly, spinal DCS, and
- Avoid excessive tissue gradients of the heavier inert gases, eg. nitrogen, which tend to give vestibular problems.

The first of these relies upon maintaining the lung as an effective bubble trap for the mass of asymptomatic bubbles (and other emboli) which decompression can produce in the venous system - largely derived from fatty tissues. Our research on the lung is still at an early stage, but bubble filtering action seems to be impaired by:

- 1. Poisoning of the lung by excessive O_2 for too long a period. This implies conservative use of oxygen for prevention and treatment.
- 2. Contaminants in the breathing mix.
- 3. Overload of the lungs by bubbles. This implies avoiding deep air diving where large amounts of nitrogen can be

liberated from fatty tissues in which this gas is most soluble.

4. Recompression. This suggests giving the minimum recompression to a diver with limb bends for fear of permitting trapped bubbles to enter the arterial system and produce a CNS "hit".

On the last point, there have been several cases of asymptomatic divers accompanying a colleague to pressure who has a limb bend, only to develop CNS symptoms themselves.

Vestibular problems

Vestibular problems can occur *without* decompression if there are large gradients of the heavier gases. The mechanisms which have been proposed include:

- 1. counterdiffusion supersaturation,
- 2. gas-induced osmosis, and
- 3. counterperfusion supersaturation

The first is unlikely since the mechanism requires a lipid layer of appreciable thickness and there is no such diffusion barrier in the inner ear. However, whichever mechanism predominates, all are realities in some tissue and would act in the same sense in producing local pressure differentials to insult the vestibular apparatus. According to each, it would seem advisable to avoid excessive gradients of heavy gases by such means as:

- 1. Adding a little nitrogen to the diving mixes if the subject is going to switch to air upon transfer from the diving bell to the DDC.
- 2. In going to 500 feet for example, compressing part of the way, say to 100 feet, on air to force some nitrogen into the middle ear.
- 3. Slowly venting the bell with air before transferring the divers to the DDC.

Breathing mixture

So far we have only considered the relationship between depth and time in programming the decompression - with decompression sickness as the sole constraint. We really need a simultaneous optimisation of depth *vs* time *vs* oxygen fraction in which there is a further constraint contributed by oxygen poisoning.

However this requires the limits for oxygen poisoning to be expressed quantitatively - not just for a constant inspired $PSDO5(O_2)$ as quoted in the USN manual, but for a complex oxygen history so common in diving. Two methods have been proposed:

- 1. That based upon the total UPTDs (unit of pulmonary toxicity dose) based upon changes in vital capacity of the lung. This gives some "handle" on the maximum oxygen which the lung can tolerate over long periods but is rather restrictive in optimising since it cannot allow for the known regression of the insult upon return to a sub-toxic mix.
- 2. A cumulative oxygen toxicity index (COTi) aimed primarily at predicting the onset of neurologic symptoms of O_2 poisoning for which there are no reliable warning signals until it is too late to prevent. This index is based upon the principle of superposition (Figure 14) which seems to hold to within \pm 10% in animals and men.

The second approach allows for regression of the oxygen insult upon reversion to a non-toxic breathing mixture and has been used for a simultaneous optimisation of depth vs time vs breathing mixture which is not so difficult to implement if a computer is available.



Figure 14 Simple arithmetic basis for the Cumulative Oxygen Toxicity Index - from Hills (1976).

"IS IT SAFE FOR WOMEN TO SCUBA DIVE DURING PREGNANCY??"

If a woman asks ten different obstetricians this question, she will probably get ten different answers, none of which are substantiated with case histories of pregnant scuba divers. Factual information on the effects of scuba diving on the pregnant woman and the foetus is practically non-existent. Opinions which have been offered by diving physiologists are contradictory and based upon studies of rats breathing hyperbaric oxygen and Japanese breath-hold divers. To date, no research has been published on any studies of pregnant scuba divers.

Margie Bolton, a graduate student in nursing at the University of Florida and a senior advanced diver, is conducting a survey to collect information from women who have been pregnant since scuba certification and within the last five years. Women who dive prior to, but not at any time during pregnancy, for the purpose of describing and comparing diving and obstetric histories.

Questionnaires will be available 1 June 1978. Results of the study will be mailed to all participants and will be submitted for publication in diving and medical journals to better inform the diving public and medical personnel of the findings of the study.

If you know of someone who would be interested in participating in the study, if you would like more information or would like to help by distributing advertising handouts to your local dive shops, instructors, dive clubs and obstetricians, please contact:

Margie E Bolton 3311 NW 30th Ave Gainesville, FL 32605

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NO SEX ON THE RIG

Ms Jennett O'Keefe, associate to WA's only Commonwealth Conciliation and Arbitration Commissioner (Mr Jim Coleman), was forced to stay ashore recently when the commissioner, employer, and union representatives inspected working conditions on the offshore drilling rig Regional Endeavour. The owners, Atwood Oceanic, refused to let her aboard because of her sex This followed a week of arguments against Ms O'Keefe's presence by the company, according to Mr Boronovskis (secretary of the Merchant Service Guild). "I was absolutely amazed.", he said, "They even used the separate toilet argument". It was claimed that the employers wished to spare her the possibility of seeing a naked man, dress standards being casual on tropical offshore rigs. As a clincher it was suggested that her presence would so distract the men that she would impair safety procedures. It was predicted that if there was a sudden emergency the workers would all abandon their duties to rescue the woman, leaving the rig to its doom.

Mr Boronovskis raised the point that at a time when they are training women astronauts for the moon it is hard to believe that they cannot be allowed to visit an oil rig. This brought the witty response that "they send monkeys to the moon, too".

This report (<u>Australian</u>, 11 February 1978) casts a new light onto the prevention of rig accidents. The too-gentlemanly rig crew should be replaced by the tougher minded management staff. They would seem, however, to be eminently suitable candidates for positions in the PR department of the firm!

EVACUATION OF DIVERS UNDER PRESSURE

Commander SA Warner - Chief Inspector of Diving, Department of Energy, London

For several years discussions have centred around the action that should be taken to safeguard divers who are in saturation or in long diving schedules when an evacuation situation arises.

Great care has been taken when studying past discussions, that the view presented was not distorted by emotion. Nobody will deny that the thought of divers being locked in a chamber and unable to assist themselves in the event of a blow-out, fire, collision, etc. presents a horrifying picture. However, it is estimated that considerably less than 200 men at peak period, are at possible risk in the whole of the Northern European area (probably less than 100 in the UK sector). In many cases these men are on board a ship which has the mobility to get itself out of trouble under some sets of circumstances.

There is no one system which will cater for every eventuality and considerable care must be taken to ensure that badly thought out and quickly introduced "good ideas" do not put men at more risk by introducing additional hazards.

In order to meet the criteria of "providing every reasonable practicable safety measure" the following conclusions from studies to date have been drawn up.

History

Throughout the entire world history of the offshore industry, on only one occasion was it considered necessary to evacuate divers under pressure. In actual fact, even in this solitary case, the divers would have been safer had they remained on board. In many other cases studied, in which divers were not involved, premature evacuation resulted in unnecessary casualties.

Prevention

It cannot be stated too often that the response to an emergency situation will never be as effective as prevention of the situation. There are undoubtedly certain times in offshore operations when the risk may be higher. There are also certain times when the risk of collision to a vessel is higher. Already 500 metre safety zones are established around installations to provide additional safety.

The UKOOA Diving Committee in discussion with the AODC have accepted an invitation from the Department of Energy to produce guidance on the subject of when, if possible, divers should not be under pressure.

It goes without saying that the highest standard of collision prevention, fire prevention, damage control and fire fighting systems are essential.

Airborne Transfer

With the introduction of the airborne system for diver casualty transfer there is an important "spin-off" safety factor, in that, the system can be adapted for a total evacuation transfer under pressure of up to eight men. A helicopter transfer to the nearest compatible chamber is a safe and comparatively simple exercise.

Crane Transfer

With the introduction of safety vessels in each small area of the North Sea facilities for crane transfer of a compression chamber (part of a compression chamber complex)

should be made. The safety vessel should be capable of providing the essential lift and the life support services for the chamber once it has been transferred.

Pressure Chamber committed to the Sea (Hyperbaric Lifeboat)

It has been argued that every person in a ship or installation should have the facility of a seat in a lifeboat in the event of an emergency, and it has also been argued that passengers in aircraft are not issued with parachutes. However, a diver under pressure requires very much more than basic survival equipment, and the cost of producing a safe floating chamber with all the necessary life support systems is extremely high.

It is possible and indeed probable, that the deployment of a "hyperbaric lifeboat" would subject the divers to an even increased risk.

Premature Evacuation

With the airborne transfer or the crane transfer system a premature evacuation of diving personnel to an adjacent rig, ship or even to shore would not put the divers at increased risk. However, the premature deployment of a hyperbaric lifeboat could be dangerous.

Early evacuation by air or crane transfer is to be encouraged, but early evacuation by hyperbaric lifeboat could introduce greater danger. However, evacuation by hyperbaric lifeboat has to be considered and acted on very early in an emergency situation which could lead to the chamber being committed to the sea unnecessarily.

Conclusion

With the state of the art today it is considered that the application of prevention: backed up by a "fly-away" capability and a "lift-off" capability fills the requirement of providing "every reasonable practicable precaution". The recommendations of the UKODA Committee on the applications of preventative techniques will be circulated as soon as they are available.

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PROJECT SEAFARER RATED SAFE

Since its conception, Project Seafarer, the huge underground antenna grid system proposed by the US Navy for communicating with submarines, has been controversial. President Carter considers Seafarer to be essential to national security. Other persons fear that the extremely low-frequency radio waves to be used could cause biological damage to people (specifically, increased serum triglyceride levels), orientative and navigational problems for birds, and behaviourable difficulties for fishes.

A National Academy of Sciences (NAS) committee has now evaluated preliminary studies of potential effects and has concluded that it is "very unlikely" that people living near the Seafarer system, if it is constructed, would be adversely affected by it. The committee did recommend, however, long-term studies of certain biologicalecological aspects to obtain more definitive information.

> (Reproduced from <u>Sea Secrets</u>, a publication of the International Oceanographic Foundation (vol 21, 1977), to whom our thanks are due.)

FREE ASCENTS: A VIEW FROM THE SCOTTISH SUB-AQUA CLUB Professor ASG Curtis President, Scottish Sub-Aqua Club

I have been asked by Dr D Walker to write a defence of the practice of free ascents in the training used by the Scottish Sub-Aqua Club.

Speaking purely personally for a paragraph, I would remark that I have been surprised at the intense and righteous disapproval that some people have evinced on hearing about our use of free ascents. It seems to me that as soon as we have a perfect knowledge about how we should dive and train it will be time to start throwing stones at those who are clearly amongst the imperfect. We are not at that happy state yet and the SSAC regards its present practices as the best it knows, but is quite aware that in the future both the accumulation of evidence and of thought is bound to alter at least some if not all of our training. Thus the views expressed in this article represent the present and the historical position but cannot be held to be a statement of what we may do in the future.

First, what do we do? SSAC training and testing is not greatly different from CMAS or even from BSAC training. The stages which a trainee should pass through are Snorkel Diver, 3rd Class Diver, 2nd Class Diver, and then onwards to 1st Class Diver and/ or a variety of instructional qualifications. During the open water tests for the 2nd Class award the trainee, who by now will have at least twenty and probably more than thirty open water dives to his or her credit, is asked to complete, after appropriate training, a test in which a slow (1 metre per 3 seconds) free ascent in a non-buoyant state from 6 or 7 metres depth to the surface is performed.

Why do we do it? Basically for three reasons:

- 1. Free ascent situations will occur, however careful we are about matters like equipment servicing, dive planning, and avoidance of situations which might lead to free ascent. After all, a great deal of training is directed towards situations which never ought to happen, eg. rescuing someone else. In recent years, as well as earlier, at least 16 incidents per 10,000 dives have occurred in the SSAC in which such events as equipment failure, air supply exhaustion, rescue of panicking divers, and very occasionally unforeseen difficulties in handling potentially buoyant articles on wrecks, etc. have led to the necessity of making free ascents. Similar incidents have occurred in other clubs.
- 2. Our training philosophy is that training is mainly towards meeting potential emergencies and that it should be practical rather than purely theoretical. In other words it is better to have some practical experience of one's ability to cope with a potential emergency situation (simulated in training) rather than a purely theoretical knowledge, as this gives greater insight and confidence as well as proven ability: provided that the risk in training is appreciably smaller than the risk in not being practically trained. We should also look at the likelihood of a situation arising and thus determine whether the training should be given to every diver or only to those who have both more experience and more probability of carrying out a large number of dives and thus of encountering the situation. We also need to analyse each situation and the appropriate response(s) and consider at what point, taking into account other practical and theoretical knowledge required, should the training be introduced.

3. At this point we enter an area where information is partially lacking. Nevertheless it is clear that practicing free ascents as we do it (see below) must have a fairly small risk. The Scottish Sub-Aqua Club has now completed more than 2800 free ascents without incident since the practice was reintroduced. It can be argued that perhaps a very small amount of barotrauma might have been detectable shortly after the ascents but there is no evidence from which to suppose that this was so.

The Club is in the process of introducing a regular requirement for repeat medical examinations for divers and those, admittedly few, divers who have been re-examined after undertaking free ascents have shown sign of lung damage.

Are free ascent accidents more frequent in those clubs that ban their practice? We do not know as yet, but we do know that incidents requiring free ascent do occur during dives. Evidence on the incidence of various types of accident in the SSAC is set out in Table 1. The data for this table was collected in a recent survey.

Basically, the table tells us that air failure is a far commoner incident than decompression sickness, or hypothermia, or unexpected sickness unconnected with diving, manifesting itself during a dive.

It is worth looking at the events and arguments that led the RN to suggest that free ascent training should be banned. In the late 60's and early 70's RN had a small number of cases of fatal barotrauma during submarine escape training, mainly amongst the trainees. The training requires the use of a very fast highly buoyant ascent with speed of 2 metres per second or faster with buoyancy in excess of 10 kilograms. Clearly these very fast ascents do have a relatively high risk of barotrauma, perhaps particularly amongst those who have little or no previous experience of being underwater. It can be pointed out that the nearest equivalent situation for the amateur diver arises either during ABLJ training, in which mismanagement can lead to highly buoyant ascents, or when weight belts are lost, particularly by those who have inflated dry suits or who carry a great deal of weight. If free ascent poses a great risk to the amateur diver we should perhaps consider banning ABLJ training or the use of inflatable dry suits.

Surprisingly the RN does not have appeared to have carried out any detailed research into the incidence of free ascent barotrauma amongst amateur divers in arriving at its recommendations and may not have been aware of the type of training that was in fact being used. In the SSAC the emphasis is first that would-be divers should receive whole plate X-ray examination to ensure that they are free from bullae. When practical training starts the trainee starts working on a shot line with great care being taken to ensure that he or she is very close to neutral buoyancy. Initially the trainee is accustomed to use a shot line for ascent, finning to ascend. Then the trainee works on the line ascending with his mouthpiece out, but close to hand should the need for it be felt, breathing out and with an instructor by his side. When free ascents can be done from 6-7 metres at the correct speed on the shot line, the trainee then repeats this free from the shot. Free ascents from depths in excess of 7 metres do not form part of SSAC training.

Thus though we recognise that free ascent does carry some potential risk there is a very low risk of consequent barotrauma, so low that in fact it has not been seen in SSAC. All training and diving procedures carry a measure of risk: for instance there have been at least three cases of incipient drowning in pool during SSAC training, happily obviated by watchful and knowledgeable instructors. But it is clear that the incidence of the need to carry out free ascents is very much higher, however avoidable they might have been in a more perfect world. However the SSAC regards free ascent as the solution of last remedy to an air supply failure, which should be solved by preferably making use of a companion's octopus rig, then by breathing from an ABLJ, then by a shared ascent and, as a last resort, by free ascent.

The evidence we have allows us to answer two of the three important questions which follow, and which sum up the whole question.

Do situations leading to free ascent occur with sufficient frequency to require training for this form of ascent? An incidence of one free ascent per 243 dives (about 6 years diving for the average SSAC member) suggests to us that since free ascent is the fifth most common diving incident it is well worth training for it, provided that the answer to the next question is suitable.

Is there an appreciable incidence of risk in free ascent training? The answer to this is that since no incident has occurred during the 2800-odd training ascents it must probably be a very low risk.

At this point all we can conclude is that there is little risk in free ascent training and that it trains for a fairly frequent incident. However it might be the case that trained divers who have not received free ascent training also cope just as well with free ascents as those who are trained, so we need to ask: Is there appreciable risk in not being trained for free ascent?

The SSAC cannot answer this, but we await data from other clubs which do not carry out free ascent training with interest. It should be remembered that the reasons which lead a particular diver to choose to carry out a free ascent may represent failure of reasonable maintenance of equipment, pre- or in-dive checks, misjudgement of situations and incorrect thought at the moment of accident, all of which can be reduced in incidence by better training, but that it is impossible to eliminate such human failings entirely. We plan to carry out a larger, more thorough survey amongst our members to discover if the frequency of free ascent is related to duration of experience, and whether it is commoner in our earlier trainees than in our most recent trainees.

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TABLE 1

SURVEY ON FREQUENCY OF VARIOUS TYPES OF DIVING INCIDENT

Sample: SSAC members. 4868 dives, representing 148 years of diving experience.

SHARED ASCENTS	1	incident	per	173.8 dives
RESCUE OF DIVER STARTING UNDERWATER	1	incident	per	187.0 dives
(including ascents in which rescuee				
did not use own main air supply)				
RESCUE OF DIVER STARTING ON SURFACE	1	incident	per	202.0 dives
FAILURE OF AIR SUPPLY	1	incident	per	206.7 dives
FREE ASCENTS	1	incident	per	243.4 dives
CONTAMINATED AIR SUPPLY	1	incident	per	270.2 dives
ABLJ ASCENT USED	1	incident	per	486.8 dives
OCTOPUS ASCENT USED	1	incident	per	486.8 dives
HYPOTHERMIA	1	incident	per	811.3 dives
ILLNESS NOT CAUSED BY DIVING BUT MANIFESTING				
ITSELF UNEXPECTEDLY DURING DIVE	1	incident	per	2434.0 dives
DECOMPRESSION SICKNESS	1	incident	per	170,000* dives
(Not based on sample but on whole club dat	a)			

* Data rounded to nearest 000.

Note that data under some categories may appear also in other categories. For example failure of air supply was the main, but not the only, cause of shared, free and other ascents, contaminated air supply being the other main reason for shared, free and other ascents.

DROWNING, A CASE OF "LOCAL RULES"

Maoris have claimed a curse, in retribution for damage done to sacred places, was responsible for the drowning of eight people off the Waikato coast of New Zealand recently. A local leader said "The drownings will continue as long as the land (in which a friendly water spirit, a Taniwha, lives) is in other hands". Another Maori leader points out that no Maori had ever been drowned in the area. The curse was imposed last year and since then the local council and building contractors have had a series of mishaps while trying to build on the disputed land.

Australian, 13 January 1978

FREE ASCENT TRAINING Dr John Knight

I have been asked to contribute an article on Free Ascent giving the RAN view. I cannot give an "official" view but offer my personal interpretations of current RAN practice and the reasons for that practice.

Because of past fatalities, the RAN attitude is that Free Ascent training can only be carried out adjacent to a chamber.

The RAN considers that, although with proper diving procedures there should be no need for free ascents, a properly trained diver should know how to do one and keep in practice. In fact RAN free ascent training is buoyant ascent as apparatus and weights are ditched and the sailor is helped up by the buoyancy of his wet suit. The ascent rate aimed at is the standard 60 feet per minute.

Some years ago there were deaths during free ascent training. At least one man died while being carried unconscious along the jetty to the recompression chamber. Following this free ascent training was only carried out when a portable recompression chamber, with a medical officer standing beside it, was on the jetty at the point where the trainees would surface. Unfortunately the portable recompression chambers are one man deck decompression chambers designed for the uncomplicated decompression of a fit diver. The decompression technique they were designed for involves the diver in an ascent (at 60 feet a minute) to the surface, immediate entry into the deck decompression chamber and immediate pressurization to 10 metres deeper than the depth of his first stop. He must reach this pressure within five minutes of leaving the bottom. After five minutes at this depth decompression is carried out as for a bottom time of 10 minutes longer than it actually was. For what they were designed to do, these chambers are excellent. But they are not treatment chambers.

They can hold two men but the second has to lie beside or on top of the first and cannot act as an efficient attendant. There is no room for any resuscitation. Once the patient is inside there is no way that anyone can get at him. If he vomits and inhales his vomit the chamber becomes his coffin. A further drawback is that the one man deck decompression chambers were made over 20 years ago when the idea of mating small and large chambers for transfer under pressure was unthought. So there is no way of transferring the patient to the larger RAN chamber where he can have an attendant and be resuscitated if necessary. The RAN is obtaining new chambers, both fixed and portable, with transfer under pressure capabilities which will allow for immediate treatment at the jetty edge and transfer to the larger chamber.

Recently the RAN appears to have reduced compressed air free ascent training and taken to training in free ascent using oxygen breathing apparatus. While this still puts the lungs at risk the embolus is inherently less dangerous as the oxygen will all be metabolised and the bubble will disappear in the process allowing restoration of blood flow. The problems of the single man chamber have probably played a part in this decision. The current practice is to have a medical sailor with resuscitation equipment and a stretcher at the site of training, on a jetty close to the large recompression chamber. The training party is large enough to provide at least four stretcher bearers. A doctor is in attendance at the School of Underwater Medicine, less than twenty feet from the chamber. While this system does increase the delay in recompressing the man by a minute, the pay-off is better care under pressure. <u>ASCENTS</u> Dr Glen Egstron University of California, Los Angeles

Ascents following the breathing of a compressed gas have been a major subject in every Basic, Advanced and Instructors course since the inception of diving instruction. As a result of whatever information was given, literally millions of safe ascents have been made by the diver involved in the programs. All concerned have accepted the <u>fact</u> that overpressure of the lung on ascent can result in damage which might become life threatening. As the sport has become more sophisticated we have seen a greater attention to the details and possible consequences of inappropriate behaviour under nearly all conditions of participation with the gear. It is not at all uncommon to recognize that the more one knows about something the more that person recognizes the enormity of the remaining unknowns. The more we learn about ascents the more complicated are the answers to questions about ascents. Today I believe we are somewhat victimized by knowing a great deal and trying to provide ultimate protection in an area where the mechanically perfect solution will always be subject to the variables of human behaviour.

In my understanding of the problem I must say that I cannot foresee <u>any</u> solution to the problem of ascending after breathing a compressed gas which will be completely satisfactory if our goal is ultimate protection. In any systematic attempt to reach "the" solution we will be faced with the knowledge that it will not provide for all eventualities. We will be forced to consider "trade offs" which will hopefully put the risk-benefit ratio into an acceptable framework. At this point I am forced to point out that, to my knowledge, there have been no evaluations statistical or logical which have developed an accident rate for any of the emergency procedures in our sport. We are told of "increases" in incidence without any information pertaining to the level of incidence for activity. Our recent exercise in legislation has shown us the dangers of using only "failure" data in assessing risk.

I would submit that our practice of accepting or rejecting a course of action in emergency procedures in general should be based upon an objective assessment of risk vs benefit based upon actuarial data; or lacking such data, at least look at the number of known problems against the background of estimates of participation based upon data such as certifications, Skin Diver projections or other reasonable data base.

The following positions regarding this problem should be recognized as comparative and not definitive. I do not believe sufficient data has been accumulated to take a complete position.

Ascents can be identified as normal, in which case the diver is required to exhale and ascent at a rate which will not cause a pressure differential great enough to cause damage or abnormal in which the basic constraints are the same. It would appear that our concern should be directed at maintaining a safe pressure gradient regardless of any procedural choices. <u>How</u> we maintain this "safe" gradient under our selected procedural variations becomes an important issue.

These procedural variations each have some rather apparent strengths and weaknesses.

"Normal" ascent - This practice pre-supposes that no gas trapping circumstances are present and that the rate of ascent is compatible with the exhalation phase so that a minimal pressure differential is present.

- 1. We have no requirement to assure that even beginners are checked for the absence of gas trapping defects in their airways.
- 2. There is little training in the matter of safe ascent rate. Admonitions such as "don't ascent faster than the small bubbles" are given with little reinforcement.
- 3. The checks to insure that divers "always exhale while ascending" is apparently effective. The overwhelming majority of divers look up, exhale and ascend slowly in a safe manner.

"Abnormal" ascent - This practice is undertaken in circumstances where an intervening variable resulting in stress enters the picture. Low tank pressure, equipment malfunction, loss of buddy contact, concern for personal safety, etc. are a few examples.

- 1. The risk appears to stem from a loss of self control resulting in a too rapid ascent rate. The crux of the problem appears to be the development of enough self control and relaxation to insure that the diver will not permit a significant pressure gradient to develop during the resolution of the problem.
- 2. Any technique which is used will ultimately depend upon self-control and an effective level of training.
- 3. What we should first address ourselves to is the question of teaching <u>safe</u> <u>ascents, whether</u> normal or abnormal. If venting is the problem we must teach them to vent effectively, if ascent rate is the problem we must train for slower ascent rates.
- 4. All alternative emergency procedures must be standardized, overlearned and reinforced. I suspect that much of the stress involved in using any of the emergency procedures is a result of a lack of confidence in the divers ability to perform adequately.

Questions

- 1. Do we have a data base to deal with the problem objectively?
- 2. Are there standardized procedures for
 - a. low tank pressure and related problems?
 - b. buddy breathing?
 - c. use of the auxilliary 2nd stage?
- 3. Will either the single or dual second stage system operate effectively under all conditions?
 - a. deep water
 - b. low tank pressure
 - c. two heavy breathers
 - d. cold water
- 4. Does the suggested procedure create more problems than it solves?

My investigations strongly suggest that the answer to all of the above questions is NO! Thus it appears that the evaluation of any procedure should be responsive to the question "Would the procedure be safe and effective if it were overlearned and reinforced to the point where stress was minimized?

JOINT MEETING OF PDAA AND SPUMS, Melbourne, March 1978

The Professional Divers' Association of Australasia, in conjunction with the Melbourne members of SPUMS, provided an outstanding meeting dealing with a wide range of diving related matters. This was followed by much discussion over a very pleasant meal provided through the kindness of the PDAA. Pride of place rightly went to Dr David Youngblood, the medical troubleshooter for Oceaneering International. His talk illustrated scope and the responsibility enjoyed (the word seems appropriate for Dr Youngblood) by someone in his position of overall responsibility for protecting both the divers and The Company in that order) from dangers known and those still barely He obviously believes that present expenditure on safety will be suspected. financially correct in the longterm (else he wouldn't be employed for long!) and ethically correct at all times. And he enlarged the old cry "Don't forget the Diver" to include the topside personnel whose comfort, and therefore efficiency, may too readily be forgotten. The days of demanding a tough disregard for comfort and risk are no longer in his calendar, though he has apparently "been there" on a number of tricky and important moments of diving history. He exudes a quiet air of intense knowledge and of having a very real desire to improve the safety and care of divers. He did not spell it out, but it was obvious that he reckoned that money "saved" by reducing safety would be but a small start towards the ultimate cost of the subsequent disasters. I would trust him "topside" anytime.

The problems he spoke about were those of saturation diving (the deconditioning effects on the cardio-vascular and musculo-skeletal systems, the changes in water balance, electrolytes and blood factors, HPNS, Cicardian rhythm changes, psychoadaptation and prevention of oxygen toxicity on the lung), the importance of the Diving Supervisor and the introduction of rig paramedics. He told the amusing (sic) story of the great job performed by such paramedics on an injured worker when shore physicians refused to risk their futures at the hands of legal profession, the employing Company, of the victim but not of the medics, then sued them! Yes, sued for saving a life. He also mentioned the need to maintain skills, for divers spend much time waiting, little time actually at work. !t was suggested that the paramedic would also be a diver and one of the saturation team so that in case of an emergency he would be there, already acclimatised, ready for immediate action.

Mr. Pat Washington, Oceaneering, then spoke regarding the Diving Supervisor and the need for the doctor to recognise that <u>he</u> is the "outsider" and very much in need of the help the diving team can offer. They can alert him to significant aspects of the dive ... or obey the doctor and leave him to flounder on alone. Communications between rig and shore are, in general, very poor in quality and often worsened by requiring relay through a chain of people. This introduces added problems to diagnosis and treatment. Travel by the doctor to the site and subsequent compression to chamber pressure take time, and produce fatigue factors in the doctor. It was repeated that one should NEVER GIVE MORPHIA in such situations. Trends towards 1 ATA diving methods will remove most problems, or so it is expected.

Dr Geoff MacFarlane talked about the Otitis Externa problems of Saturation Diving, something that has caused the curtailment of some diving operations ... though not any undertaken by Oceaneering. It was apparent that quite a bit of explaining would need to be done before divers would be happy to miss a high-pay saturations dive on the say-so of pathogens in an ear swab. Another major paper was by Mr Don Macdonald, Federal Secretary of the PDAA. He gave a detailed resume of the history of attempts to set up a central register containing details of diving medical examinations in a confidential but researchable manner. It seems that everyone, but everyone, is in favour of the idea, but As you will guess, somehow nothing has eventuated just yet. One can only speculate at the result had people opposed the idea!

"Ah, well, such is life," as someone once said in Melbourne.

The Official papers concluded with a brief review by Dr Ian Unsworth of Air Embolism, capped by an incident related by Dr Youngblood of the diver who was taking off his flipper in a dive boat when he suddenly collapsed with symptoms of hemiplegia, presumably the result of an air embolism, secondary to pulmonary barotrauma incurred on the ascent. This reporter was left nodding in agreement at the amended aphorism, ascribed to an eminent French Specialist of Diving Medicine, that "Murphy was an optimist". Yet still we dive, asymptomatically in the main.

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DON'T DIVE, BABY!

Professor Graham Liggins, Head of the Department of Obstetrics at Auckland University, believes that Antarctic seals may hold the key to the riddle of cot deaths. He has spent two months in the Antarctic last year studying their diving reflexes. These are the result of the seals stopping breathing and submerging and involve the cutting off of the circulation of blood to all parts of the body except the vital organs such as heart and brain. He believes that the most convincing theory on cot deaths is that the victims have overactive "diving" reflexes.

This could be triggered by water being thrown in a human's face or from certain liquids suddenly hitting the back of the throat. This reflex stops the circulation to most of the body, including the lungs: and this stops all breathing.

The Professor has said that cot deaths result from a particularly sensitive reflex being triggered by regurgitation of stomach contents against the back of the throat. The reflex would be particularly severe if the stomach contents were acidic. He hopes that his research into the breathing patterns and diving reflexes of seals will help in identifying babies at greatest risk. These included babies with minor illnesses (who are more likely to regurgitate), those fed commercial baby food (which is more acidic than human milk), and those who had already survived a previous "diving reflex". He has suggested that if high risk babies could be identified, a monitor could be placed in the cot to sound an alarm if breathing stopped.

Daily Telegraph, 21 April 1978

ABSTRACT

Women make better divers both physically and emotionally than men. Why are there no more women divers? There are problems, unique to women, that they have to overcome to eventually feel comfortable in scuba. By understanding and becoming aware of these, instructors can use them to the students' best advantage.

Women are better divers than men! We have all heard this statement, but do you believe it? Several facts support it. The average woman breathes between 0.6 and 0.9 cubic feet per minute (cfm) at the surface while the average man uses in excess of 1.0 cfm. The layer of subcutaneous adipose tissue not only insulates her efficiently but increases her buoyancy. Are these reasons really important for making women better divers. I say no. Women have calmer nerves, perform more efficiently under stress, and are more cautious. These go together in making them better divers. Since women are both physiologically and emotionally well suited for diving, why do they comprise only 20% of all divers certified by NAUI?

For years scuba diving has been male dominated. Diving used to be visualized as macho, difficult and exhausting. As the image of diving changes to one of fun and excitement, more and more women are coming into classes to learn to dive. Some have a preconceived notion of what recreational diving is all about. For the most part these students also will be easy to teach because they want to learn. Others are brought into the sport by personal pressures. These unfortunately are much harder to teach because they are not entering into sport with free minds. The fact that they all agree to take a scuba course suggests that the instructor can influence their ultimate enjoyment of diving. In order to best support woman as divers we must appreciate their reasons for getting into diving, the problems they perceive as students, and finally the realities they face after they are certified.

I wish that I could say that all woman go into diving for their own personal satisfaction. Unfortunately, this is too often not the cage. One of the of the most prevalent reasons for a woman taking a scuba course is that her spouse/boyfriend/ lover "pushes" her into it. Frequently the "push" is subtle, being left at home or on the beach once too often. Sometimes, constant nagging on the part of the male member of the pair causes her to agree to dive to shut him up. In either case, their motivation is not sufficient to allow for an easy transition from an uncomfortable novice to a competent diver visiting the aquatic environment on a regular basis. These are the women we frequently get as students, and with whom we must work, in order to increase their self motivation and ease their transition. The instructors most successful at this have learned to treat women as individuals while still being sensitive to their particular reasons for diving.

One big problem that many women have, that they cannot do much about, is their size. These pint sized divers are trying to manage equipment that is much too large. This is frustrating enough for the women that are strong and are not having problems. Think how it is for the ones that are just a little nervous and any minor hassle is a major issue.

Would you put on a wetsuit that fit your arms but was a half size too large in the chest? Would you put on a back-pack that did not fit the contour of your back? How about a tank that constantly hits you in the back of your knees? Of course not, and yet this is what the diving equipment manufacturers are asking women to do.

How many of you have ever met a women who looks like the wet suits we see hanging in the shops. A 44" bustline matched to a 5'3" body? It would be much more reasonable if the wetsuits were built to more realistically reflect the size and shape of today's women.

We all realize that getting into a wetsuit can be a struggle. It becomes real work when your hips are 10 inches larger than your waist. A simple zipper in the side of a pair of Farmer Johns readily solves this particular problem.

Put any standard buoyancy compensator on most women and it hangs down below her waist. How can they be comfortable when their BC takes up half their body length and sticks way out on either side of their bodies. Several manufacturers have come out with "shortie" BC's and these prove to be satisfactory if they are used.

Below the BC is that plethora of buckles - BC, backpack, and weight belt. In an area that usually will take only one buckle, we put all three. There are several solutions to this problem. Back BC systems incorporating weights solve this dilemma, but introduces a new set of difficulties, mainly in the back, when the woman is out of the water. Smaller webbing with half sized buckles or velcro closures might prove very effective at uncluttering the woman's midsection.

These are some of the equipment hassles that the women entering diving must face. The instructor must be aware that many apparent skill problems may just be simply manifestations of poorly fitting gear. These will largely disappear when the members of DEMA recognise the purchasing power of women divers and manufacture gear suited to their needs.

Other than the gear, women have a few more strikes against them when they decide to go into diving. This is their physiological makeup. Women have been raised in an emotional environment which enhances sensitivity and suppresses competition. This is, in large, the opposite of the cultural training a man receives. Recognizing this, is it fair for us to "hurry to the dive site", to "rush getting geared up", or to introduce "competitive games" into our training programs?

Almost anyone finding themselves competing in diving and not doing so well will feel put down. Repeated frustrations of this type go a long way in causing women to drop out of diving before they ever really get into it. Games that are non-competitive or that put "teams" against one another in fun are both enjoyable and enhance learning. The games that have a "winner" or that have some degree of failure are detrimental to the sport. Diving is noncompetitive. It is a sharing sport with each person sharing their experiences with the other.

As instructors we make a firm commitment to teach to the needs of our students. We must recognize that each person is an individual and treat them accordingly. Along with this it must be recognized that the women in our classes have their own unique problems with gear, with their buddies, and with themselves. We are dealing with a special group of people, that, if we let them, will help revolutionalize the sport into one that is truly exciting, sensual, and fun for everyone.

INSTRUCTOR OPINIONS: RESULTS OF A NAUI SURVEY Neal Langerman and Pat McIlvaine

ABSTRACT

NAUI is a member run organization which requires continual input of ideas and opinions in order to grow. A questionnaire was mailed to all meanders in April 1977, designed to sample opinions on a variety of issues including equipment, training techniques, cardiopulmonary resuscitation, and diver recertification. The responses were analyzed for the percentage of "YES", "NO", or "UNDECIDED" answers to each question. Responses to several questions allow a definitive statement to be made concerning the issues.

During the fall of 1976, an unsolicited survey was sent to NAUI headquarters by the authors of this report with the request that it be sent to all members of NAUI. The questionnaire, which was mailed in April 1977 to 3200 members of NAUI, was designed to obtain information concerning three specific areas pertaining to diving instruction, equipment, teaching methods, and diver recertification. More than 600 responses were received by 10 May (10 days after the published "deadline") and an additional 50 during the next two months. Those responses and our interpretation of the data is the subject of this report.

Completed questionnaires were returned from 44 states as well as the Bahamas, Puerto Rico, the Virgin Islands, Canada, Singapore, Guam, and Palau. The geographic distribution of the responses is presented in Figure 1. The numbers represent the percent responses received of the total members of NAUI listed for that region in the 1976 NAUI Directory. Southern California was taken to include Fresno and points south.

We have also examined the responses in terms of the distribution of the NAUI numbers of the respondents. Figure 2 presents those data along with information about the approximate length of time the respondents have been members of NAUI. We feel that these data are what one might reasonably expect, that is, those instructors who have been teaching for less than 5 years are the most likely to respond.

Approximately 10% of the responses contained letters, some of them quite extensive, commenting on various aspects of the questionnaire. We have taken the liberty to quote from several of these letters and have tried to answer many of them individually.

The questions and the results of the responses are presented in Tables 1 and 2. The questions have been grouped into the three areas previously mentioned. The percentage "YES", "NO", or "UNDECIDED" to any given question is based on the number of responses to that question. Many people indicated that they did not wish to answer a particular question.

EQUIPMENT

QUESTION 1: Should all NAUI SCUBA courses, at all levels, absolutely require the use of a submersible pressure gauge?

An overwhelming number of those responding favoured the use of submersible pressure gauges (SPG). The surprising point was the number who were opposed. While some persons indicated that their opposition was to the "requirement, not the concept",

others said that they did not wish to use SPG's in open water. These people all indicated that they preferred reserve systems.

QUESTION 2: Should a NAUI <u>BASIC</u> SCUBA course require the use of a submersible pressure gauge during all training sessions in which compressed air is used, ie. both during confined and open water work?

The responses for SPG's in all situations when compressed air is used was somewhat less enthusiastic. Two basic arguments were offered for opposing this idea. The first, and most prevalent, was equipment maintenance. This argument says that students in BASIC courses are unduly hard on SPG's and that repairs or replacement will drive the cost of the course up to intolerable levels. It is our personal opinion that this is a "straw horse". If students are carefully instructed in the care and handling of gear, few SPG's will be crushed by tanks or dropped on pool decks. Indeed, proper care of shop equipment is the first step in learning proper care of personal equipment. One argument for not requiring an SPG at all times suggests that the feeling of discomfort and uncertainty which the student experiences not knowing just how much air is left reinforces the desire to always dive with one. On the other hand, always having an SPG, even in the pool, reinforces the diver's habit of constantly monitoring the air supply. Thus, the majority of the respondents feel that SPG's should absolutely be used in open water and that they should also be used in pools except when a valid teaching reason prevails.

QUESTION 3: Should NAUI require a constant reserve system (either J-valve or an equivalent) to be used in conjunction with a submersible pressure gauge?

Constant reserve systems, on the other hand, received a large vote of "no confidence". The arguments against reserve systems ranged from "they have a high failure rate" to "they are too easily breathed through or inadvertently turned on". The sonic reserve system did receive some support and has the support of staple dive boat operators in Southern California who will allow divers in the water with either an SPG or a sonic reserve. The principle argument in favour of a reserve used in conjunction with an SPG was given by Paul Tzimoulis in the May 1977, <u>Skin Diver</u> magazine.¹ He referred to the reserve as an "alarm clock", which only works if you remember to set it.

QUESTION 4: Should all NAUI instructors be required to use an "octopus rig" for all training sessions involving scuba?

The responses to the question suggesting that all instructors use octopus rigs whenever using SCUBA elicited an almost even split. Comments ranged from "they just get in the way" to "I wouldn't take students in the water without one". Several people objected on financial grounds and several for "difficulty of maintenance" reasons.

It is quite clear that additional discussion of the pros and cons of the octopus rig is required. The entire problem of octopus training during a BASIC SCUBA course will be dealt with in another section of this report.

After reviewing the responses to the questions on EQUIPMENT, it is our opinion that members of NAUI want to keep equipment simple and safe. They are willing to entertain new ideas, but only want them incorporated into our programs after they have been thoroughly tested and discussed.

TABLE 1 QUESTIONS FROM 1977 NAUI SURVEY

- Should all NAUI scuba courses, at all levels, absolutely require the use of a submersible pressure gauge?
- 2. Should a NAUI <u>BASIC</u> scuba course require the use of a submersible pressure gauge during all training sessions in which compressed air is used, ie. both during confined and open water work?
- 3. Should NAUI require a constant reserve system (either J-valve or an equivalent) to be used in conjunction with a submersible pressure gauge?
- 4. Should all NAUI instructors be required to use an "octopus rig" for all training sessions involving scuba?
- 5. Do you give "Octopus Training" in addition to standard "Buddy Breathing" training in a BASIC scuba course?
- 6. Should NAUI require "Octopus Training" as part of all scuba courses, at all levels?
- 7. Do you teach emergency ascent training in open water during <u>ADVANCED</u> scuba courses?
- 8. Do you teach emergency ascent training in confined water during <u>ADVANCED</u> scuba courses?
- 9. Do you teach emergency ascent training in open water during <u>BASIC</u> scuba courses?
- 10. Do you teach emergency ascent training in confined water during <u>BASIC</u> scuba courses?
- 11. Should NAUI require emergency ascent training and specify one prescribed training method for all scuba courses, at all levels?
- 12. Do you teach ditch and recovery during a BASIC scuba course in confined water?
- 13. Should ditch and recovery training in open water be prohibited by NAUI?
- 14. Should all active members of NAUI, instructors, assistant instructors, skin diving leaders and divemasters be required to maintain a current CPR certification (not necessarily "instructor level" training)?
- 15. Should NAUI introduce a "Diver Recertification" requirement (of a yet to be decided mechanism) by placing an expiration date on all certification cards?

COMMENT WITHHELD ...

Jamie is 4 years old and 1 metre tall. He has only recently learned to dogpaddle, but that doesn't stop him from scuba diving. Equiped with his own mask, custom-made wetsuit and small air tank he is off to the USA with his parents (who run a diving school in NSW) to negotiate for television commercials. His scuba lessons started about five months ago and his father is quoted as saying "I wouldn't try to hold him back in something like this. It is better for him to learn the correct way now than have him experiment". His deepest dive has been three metres in a training tank. Occassionally he dives in the shallows at the beach with his father close by.

Australian, 22 February 1978

INUMBER	% YES	NUMBER	% NO	NUMBER	00	TOTAL
YES		NO		UNDECIDED	UNDECIDED	NUMBER
423	72	162	28	_	_	585
386	66	201	34	_	_	587
97	17	468	83	_	_	565
285	48	248	41	65	11	598
225	66	170	34	_	—	341
172	30	347	58	76	12	595
410	75	140	25	_	—	550
433	79	116	21	_	—	549
357	60	242	40	_	—	599
535	90	59	10	_	_	594
290	48	202	34	106	18	598
541	92	50	8	_	_	591
166	28	329	55	105	17	600
407	68	129	22	64	10	600
276	46	197	33	126	21	599
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TABLE 2 RESULTS OF 1977 NAUI SURVEY

DIVING SAFETY MEMORANDUM NO 8, 1978

Commander S A Warner, Chief Inspector of Diving, Department of Energy, Petroleum Engineering Division, Millbank, London SW1P 40J

Diagnosis of Decompression Sickness

During 1977 in the United Kingdom sector of the North Sea there were several occasions when the Diving Supervisor failed to correctly diagnose decompression sickness. On three occasions, what should have been a normal therapy, eventually required a saturation type therapy. Excuses such as cramp have been used in order to explain what are in fact serious symptoms.

The section on diagnosis of decompression sickness in the United States Navy Diving Manual is drawn to the attention of all Diving Supervisors and in particular, the "patient examination":

Does diver feel well?	Yes/No
Does diver look and act normal?	Yes/No
Does diver have normal strength?	Yes/No
Are diver's sensations normal?	Yes/No
Are diver's eyes normal?	Yes/No
Are diver's reflexes normal?	Yes/No
Is diver's pulse rate normal?	Yes/No
Is diver's gait normal?	Yes/No
Is diver's hearing normal?	Yes/No
Is diver's co-ordination normal?	Yes/No
Is diver's balance normal?	Yes/No
Does the diver feel nauseated?	Yes/No

The correct application of the above table can avoid extremely serious consequences.

TEACHING METHODS

QUESTION 5: Do you give "Octopus Training" in addition to standard "Buddy Breathing" training in a BASIC SCUBA course? QUESTION 6: Should NAUI require "Octopus Training" as part of all SCUBA courses,

at all levels?

OCTOPUS TRAINING

Ten years ago, when submersible pressure gauges were considered new equipment, the question of always using an SPG would have drawn an indecisive response from instructors. Today it does not. 66% of those responding do teach "Octopus Training" in addition to standard "buddy breathing" training, but 58% oppose NAUI requiring such training in a BASIC course. It is our experience that teaching students to use an Octopus after teaching them to buddy breathe is easy. We never have any difficulty with students learning this skill. It doesn't require a large expenditure of money either; having them breathe off one of our instructor's extra second stages, both in the pool and in open water, in conjunction with a few minutes of lecture, helps prepare our students for the use of an Octopus.

As we indicated previously, the entire concept of an Octopus rig still requires debate and discussion among divers and instructors. However, if "Safety through Education" is to remain more than just a trite phrase, can we ignore this simple step toward safety?

QUESTION 7: Do you teach emergency ascent training in open water during <u>ADVANCED</u> SCUBA courses?

QUESTION 8: Do you teach emergency ascent training in confined water during ADVANCED SCUBA courses?

- QUESTION 9: Do you teach emergency ascent training in open water during <u>BASIC</u> SCUBA courses?
- QUESTION 10: Do you teach emergency ascent training in confined water during <u>BASIC</u> SCUBA courses?
- QUESTION 11: Should NAUI require emergency ascent training and specify one prescribed training method for all SCUBA courses, at all levels?

EMERGENCY ASCENT TRAINING

Certainly the issue of "Ascent Training" is the most emotional issue facing instructors today. Students, it may be argued, need the confidence that doing a controlled swimming ascent develops. The National SCUBA Training committee has just agreed² upon a set of "Emergency Procedures" for use when a diver is out of air and has nowhere to go but up. These procedures include a swimming ascent.

QUESTION 12: Do you teach ditch and recovery during a BASIC SCUBA course in confined water?

QUESTION 13: Should ditch and recovery training in open water be prohibited by NAUI?

During open water classes, 75% of NAUI instructors teach Emergency Ascents in ADVANCED courses and 60% in BASIC courses. Jon Hardy has informed the authors that very few waivers are outstanding which relieve the instructor of the obligation to teach emergency ascents in open water. In confined waters, most instructors are teaching emergency ascent procedures.

It is interesting to note that even instructors who refuse to teach swimming ascents in confined water do teach ditch and recovery. Certainly this skill has all of the same danger of cerebral air embolism associated with it as has emergency swimming ascents.

Ditch and recovery is usually taken to mean removing a SCUBA tank, leaving it on the bottom, swimming to the surface, and then diving down and donning the tank. 55% of the respondents want this to be allowed in open water. This is surprising in the light of the feelings toward emergency swimming ascents. Based on the comments related to this question, it appears that "ditch and recovery" may have been interpreted to mean the removal and replacement of a tank while remaining on the bottom.

Question 11 deserves particular attention. As many people pointed out to us, it really asks two separate questions. To the first, "Should NAUI require emergency ascent training?", a small majority said "YES". To the second, "Should NAUI specify one training method for all SCUBA courses at all levels?", an overwhelming majority said "NO". The data reported in Tables 1 and 2 represent the average of these answers.

Several conclusions may be drawn from the questions involving Emergency Ascent Training. First, the problem of definition still has not been solved. Dennis Graver³ and Jon Hardy⁴ have each explained this term and several related terms on many occasions. We suggest that you review these definitions. NAUI members want the option of teaching this skill, but they also want the freedom to teach it as local conditions dictate, or not to present it as a practical skill at all. The current waiver systems certainly satisfies these needs, but it must be used by the instructors. Finally, members of NAUI appear to feel that teaching how to perform an Emergency Swimming Ascent is a integral and important part of SCUBA training. This information, including the numbers, should be used as an argument to present to our insurance carrier's and others, if they, who DO NOT teach SCUBA themselves, try to tell us what should be taught.

QUESTION 14: Should all active members of NAUI, instructors, assistant instructors, skin diving leaders and divemasters, be required to maintain a current CPR certification (not necessarily "instructor level" training)?

<u>Cardiopulmonary Resuscitation</u>: Last year at 10_g , it was stated that CPR training need not be an integral part of the skills of an instructor nor part of a scuba course, since "it doesn't work anyway". Apparently, the members of NAUI disagree. Member opinions on CPR ranged from "it is too difficult to find an instructor to teach it" to "it is the most valuable skill we have ever learned." To the first we say "become a CPR instructor yourself" and to the latter, we say "hooray"!

CPR does work! It is not difficult to learn⁵ and takes only 3 hours for a CPR qualified instructor to teach. Bob Widmann has just pointed out in the July/August 1977 NAUI NEWS that this skill is so important that time must be made for it in SCUBA classes. It is quite apparent to us that the members of NAUI recognize this skill and want it to remain part of the NAUI program. Indeed, many feel it should be a requirement to remain on an ACTIVE status within NAUI.

DIVER RECERTIFICATION

QUESTION 15: Should NAUI introduce a "Diver Recertification" requirement (of a yet to be decided mechanism) by placing an expiration date on all certification cards?

The question of a "lifetime certification" elicited almost as much comment as that of "emergency swimming ascents". OD Wells' letter in NAUI NEWS⁶ and the several letters in response to it presented the broad spectrum of opinions. The survey indicates that the respondents to the questionnaire are split 46% to 33% (the remainder undecided), but these numbers hide some very strong opinions. It is certainly true that NAUI cannot unilaterally put an expiration date on their certification card and hope to remain a viable enterprise. It is also true, that of the three sports which require BASIC certification (SCUBA DIVING, SKY DIVING, and FLYING), only flying requires continued proof of competence and this is a Federal requirement. Finally, a recertification program runs the risk of generating diver animosity and chasing people away from the sport. On the other hand, SCUBA DIVING is a sport with a conscience - we recognize the inherent difficulties in the sport and each of us, from the equipment manufacturers through the weekend diver, accept certification as the method which prevents needless accidents and losses of life.

Will divers accept a recertification program? Will the retailer accept the onerous responsibility of trying to enforce it? How will it function? Many people responded to the last survey question with detailed answers. Some of their comments are: "recertification for someone who has been out of touch for a long time is fine, but it will be a great imposition to those who are active" (Scott Leonard); "Perhaps the log book holds the answer. BS-AC (British Sub-Aqua Club) has now for a long time used a log book instead of a certification card and their divers are proud to update or upgrade their log books" (Bob Friedman); "Diver recertification has many logistical problems. The best recertification is active diving experience Mandatory certification is not going to help the person who dives with his ego, rather than his brain." (John LeClair); "I've been in favour of this for years. A lot of co-ordination and good-will among organizations training divers and among instructors in NAUI will have to happen before we could pull it off." (Bob Landers); "I offer free tests to allow an individual to test his knowledge. I also have dives during the summer for certified but not so current divers who wish to get back into the sport." (Wayne Dykstra). Comments such as these could be continued for several pages, but the content should be apparent from these examples. Clearly, this is a subject which still requires more debate and certainly must have the co-operation of all of the training organizations.

In retrospect, we consider this survey to have been quite successful. The 20% response, which is remarkably high for this type of survey, is very encouraging. We feel we have gained considerable insight into your opinions. This information, and information gained from future surveys should help keep NAUI the quality organization which it currently is.

REFERENCES:

- 1. P Tzimoulis, Skin Diver Magazine, May 1977.
- 2. NAUI News, July/August 1977.
- 3. D Graver, 10g Proc., p 132.
- 4. J Hardy, NAUI News, March 1976.
- 5. B Widmann, NAUI News, July/August 1977.
- 6. OD Wells, NAUI News, October 1976.

<u>PADI TRAINING BULLETIN 78-1</u> Dennis Graver, National Training Director

1978 Proposed Standards Changes

The following standards changes are presented for membership consideration and comment. The revisions will be finalized, approved, and published in April. They will be effective on June 1 as usual. The proposed revisions are:

- 1. To allow the skin dive and two scuba dives for BASIC certification to be conducted on one day. Having training take place on more than one day is recommended and desirable but not required.
- 2. To modify the Student-to-Instructor ratios as follows:

Α.	Skin diving (Pool)	16:1
в.	Skin diving (Open Water)	10:1
C.	Scuba diving (Pool)	10:1
D.	Scuba diving (Open Water)	6:1
Ε.	Introductory Course (Resort Course)	4:1
F.	Divemaster Training	6:1

- 3. To limit the total number of students in an OPEN WATER training group with one Instructor and the required assistants to a maximum of 14.
- 4. To require use of buoyancy control devices in all pool scuba training sessions.
- 5. To remove the requirement to compute air consumption during training.
- 6. To require BASIC and OPEN WATER Diver students to experience running out of air in a controlled situation (pool) during training.

The membership has already indicated the need for most of these revisions. Reference the Training Revision Survey results in the JOURNAL, Vol. X, No. 5, page 13.

Suggestions regarding these revisions or other needed standards changes should be sent to the National Training Director by 1 April for consideration by the Board of Reviews.

PARROT FEVER FROM CLAMS

A research team from the Smithsonian Institution and Maryland Department of Natural Resources has been looking at marine animal diseases by studying the gut contents of Chesapeake clams and oysters under the high magnification of an electron microscope. They find shellfish infested with a variety of phages and microbes, including some that resemble the chlamydia of psittacosis, the disease of parrots that also infects humans. Thus, they suggest, clams may transmit this disease to humans who eat raw clams.

Sea Technology, June 1977

Are the days of deadliness of the shy blue-ringed octopus numbered? There is enough venom in the adult's two tiny sacs to kill 10 people. But now Macquarie University reports that a five-member research team has discovered the chemical make-up of the main lethal toxin in the venom. It is identical to the known compound, tetrodotoxin, present in toad fish, some newts and frogs. Now what is needed is the antidote. Sydney Morning Herald, 19 Nov 1977

BOOK REVIEW:

THE BELLE OF SUNDA STRAIT

by David Burchell (Rigby 1971)

This is the story of one man's successful attempt to recover portions of HMAS Perth, sunk in action in the Sunda Straits on 28 February 1942. Anyone who has ever tried to get permission to do something out of the ordinary, let alone get practical backing in cash and kind, will find David Burchell's belief that his project could succeed hard to credit. He managed to get considerable help from the Indonesian Naval Authorities, help few other countries would have offered. That they did try to tell him that solo diving with Scuba in strong currents at 230-250 feet in the open sea would be unsafe advice he somewhat patronisingly puts down to their ignorance of modern practice! The tale shows what a determined and singleminded person can accomplish, given the required personal skills and access to persons with some sympathy for his aims. Although the tale is a little short on exact dive depths and times there are a number of incidents described of the "didn't ought to have done it" type. But first find your wreck.

Yes! You get the best results by asking the local fishermen. After all it is they who lose nets on such underwater objects. He comments on the very great skill they have in fixing locations by visual bearings without, it seems, using instruments. Then one dives ... but only David Burchell (I hope), would attempt such dives. Imagine a solo descent through water opaque with algae that make the line slippery, in a current that is persistent and strong, wearing a single 72 cubic foot cylinder and the only help being a companion (non diving) with a spare set in a small dinghy. Down you go to 160 feet and hope no sharks appear. The thick wet suit made life very unpleasant before water entry but it at least protected from the coral and the sea wasps!

Pity John sitting there in the boat, probably thinking what he would tell the Coroner. Pity the helpful Indonesian Authorities fearful of a loud outcry at their allowing such a crazy diver to get in the water, let alone giving him help. As David admitted, John was never very happy when he was under the ship trying to enter the Quartermaster's lobby, a space already occupied by several large groper and one large octopus. Such a dive led to the exhaust bubbles becoming trapped so the boat "cover" had nothing to show that the diver still lived. At least in other situations the air bubbles gave comfort to those in the dinghy as they reached the surface. And on the 29th dive he was really tested. With about 5 minutes dive time air left he suddenly found himself grabbed by the back of the neck as if he was nailed to a wall. He discovered that a tangle of loose wire had fouled the regulator and his description of the problems of getting loose, without being so foolish as to loose his expensive camera, should persuade everyone of the folly of solo diving in a wreck. Like he says, it wasn't the best place to be caught.

And one footnote, for he forgets to mention it in the text, that illustrates the power of the human spirit to overcome difficulties. David lost one leg in an accident when he was 16, but he has been more active in his life than almost any dozen "intact" persons.

SUBSCRIPTIONS

Members pay \$15.00 yearly. Associate membership for those neither medically qualified nor engaged in hyperbaric nor underwater related research is available for \$10.00. The journal is sent up to four issues yearly to both full and associate members. Those resident outside the immediate Australasian area should write for the special terms available.

Treasurer: Dr W Rehfisch, 5 Allawah Avenue, Frankston VIC 3199

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NOTES TO CORRESPONDENTS AND AUTHORS

Please type all correspondence and be certain to give your name and address even though they may not be for publication. Authors are requested to be considerate of the limited facilities for the redrawing of tables, graphs or illustrations and should provide same in a presentation suitable for photo-reproduction direct. Books, journals, notices of Symposia, etc will be given consideration for notice in this journal.

Address correspondence to:

Dr Douglas Walker PO Box 120 NARRABEEN NSW 2101

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