SPUMS JOURNAL
ISSN 0813 - 1988
South Pacific Underwater Medicine Society
Vol 16 1986 No. 2 APRIL-JUNE

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Registered by Australia Post
Publication Number VBH 3527

PRINTED BY: KD COPY CENTRE
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EDITORIAL

Despite a seemingly morbid concentration on fatalities it would be wrong to subtitle this issue "Nought for your Comfort". Indeed it contains a great deal of information highly relevant to the avoidance of critical factors identified as being associated with increased risk of death or injury while diving. These factors include the health of the diver and the letter from David Wailes politely but bluntly sets out the problem faced by careful diving instructors. This problem has been ventilated in these pages on previous occasions and several writers have given advice concerning both the absolute and relative contraindications to granting a certificate of Medical Fitness for Diving. While attendance by the doctor at some Diving Medicine course cannot ensure that he has accepted the dicta of the Experts (and sometimes it is correct to have views differing from those of 'experts'), it is at least documentation of some attempt to gain knowledge. As was stated in the last issue of this Journal, there will be many with great knowledge and understanding of the medical factors effecting diving safety who have acquired their expertise through other routes. For this reason it will not be an easy matter to provide an authoritative listing. The UK system of courses of training before doctors are accepted on the list of those approved to examine professional divers has much to commend it, but even this is a scheme which ignores the larger number of recreational divers. It is likely, therefore, that real progress much await a decision of the major Instructor Organisations to become involved in this matter, remembering always that good training is undoubtedly the most important single factor influencing diving safety.

Both the New Zealand and the United States are represented by surveys of their recent diving fatalities. The former report reveals an alarming incidence of medically adverse factors in those who died, although in most instances other factors were more immediately critical. It must be a matter for concern that divers are so unaware of the potential dangers of scuba diving that they loan their equipment to friends who lack all knowledge of diving. Some of the cases point up the ethical difficulty of advising someone who has a known health hazard but who nevertheless wishes to LIVE his life. The quality of the advice which is offered will in large degree depend on obtaining input from those already diving who have 'escaped the medical net' and keep a discrete silence concerning their medical conditions. It would be of immense benefit to many if they could be persuaded to record their stories.

The American record has a wider sweep, containing a much larger number of cases as their number of active divers is greater. Once again there has been an appreciable death rate among those under instruction, this being a matter of surprise to readers who imagine the high US risk of litigation would scare instructors into being ultra cautious. Details of this cohort of divers are awaited with interest. Sudden death from heart disease also appears to be a significant item, although it is not certain that any routine medical check would identify those at risk. Australian experience supports the suggestion hat sudden death while swimming at the surface is a real danger, and use of the term "Sudden Drowning Syndrome" may increase awareness of this condition. On Australian experience it appears that sudden death is rare when close contact is maintained by the buddy. The US Navy diving tragedy will certainly be subjected to close scrutiny and it is hoped that additional details will become available later. The Florida fatality caused by a severe jellyfish envenomation is a reminder that Queensland has no monopoly of such creatures. Papers from a recent meeting in Queensland on this subject will be printed in a coming issue of the Journal.

Other papers deal with a range of subjects of importance, from Diving Research to Rehabilitation, from the Pathophysiology of Drowning to a paper on the Art of Breathing, and there are many more matters ready to interest you in the next issue of the Journal.

ORIGINAL PAPERS

PROVISIONAL REPORT ON NEW ZEALAND DIVING-RELATED FATALITIES 1983-1984

Douglas Walker

SUMMARY

During the years 1983, 1984 there were six fatalities in breath-hold divers (4,2) and fourteen (7,7) in scuba divers. The full facts are not available in many cases but sufficient details are known to indicate some major avoidable adverse factors.

The breathhold diving fatalities include demonstration of the dangers of fatigue, cold, hyperventilation, cardiac factors, asthma, previous chest disease and epilepsy. Such factors are only rarely themselves the sole critical cause of the fatal outcome, a common additional factor being separation from the buddy or the lack of a buoyancy vest. Two of the deaths were considered to be unavoidable in the particular circumstances of their occurrence (cases BH 84/1, BH 84/2) but to have a direct relationship to the stresses (fatigue, cold, effort) associated with their dive which made the critical illnesses non-survivable.

Scuba related fatalities similarly show separation and a lack of or failure to use a buoyancy vest, though an inflated vest is no guarantee of survival. Inexperience and absence of formal instruction in scuba diving are common findings, though even the best of supervision may not prevent a fatal outcome where cardiac factors operate strongly.

CASE REPORTS

The reviews are based on information given at Inquests. It should be remembered that "the Coroner's function is to establish the identity of the deceased person and when and where he died." This definition by a New Zealand Coroner accurately defines the basic function of any Inquest and it is fortuitous and welcome when the search to establish the "why" of a diving-related death documents all the details which are necessary to analyse the many factors influencing the final critical path. The Coroner's task is easier when the medical evidence is given by pathologists fully aware of diving apparatus and diving pathology. The term "asphyxia" should never be used without an explanation of how it has been produced: it is not the expected result of running out of air using scuba.

BREATH-HOLD DIVING CASES

Case BH 83/1

This 21 year old man, in company with a friend, dived for mussels on a reef 100m from shore. They started when it was low tide and the water was neck deep but an hour later the tide had come in and they could no longer touch bottom when standing. The victim was described as a strong swimmer but this was only the 3rd or 4th time he had dived for mussels. He was wearing a wet suit jacket, mask, a borrowed weight belt which did not have a proper quick release, and boots, not fins. The buddy noticed that the victim seemed to be in trouble but was himself feeling too tired to offer any help so he called to people ashore to come to give assistance and himself swam back to the shore. There is no mention of the sea conditions at the time. The body was found two days later, weight belt in position.

GROSS INEXPERIENCE. SURFACE PROBLEM. BUDDY TIRED SO LEFT VICTIM. BOOTS WORN NOT FINS. WEIGHT BELT NO QUICK RELEASE. NO SNORKEL. NEITHER DIVER WORE A BUOYANCY VEST. DIVING FOR MUSSELS.

Case BH 83/2

It was only the second time he had gone diving and he had borrowed equipment from a friend. He was an epileptic, on medication for 2 years, who had "warning twitches" before any full epileptic attack developed. He had permission to swim if he was accompanied, but "diving" had not been discussed. He and another boy snorkelled out to some buoys about 100m from the beach and there the buddy lost sight of him momentarily while ducking under a rope in the water near the buoy. When he looked again he could not see his friend but saw some bubbles breaking at the surface. He immediately dived but was unable to reach the sea bed, a depth of 15 metres. The body was recovered later by the police divers. It is assumed that he lost consciousness and drowned as a result of an epileptic fit. Possibly he failed to recognise the warning signals because of his concentration on snorkelling, a totally new experience. The buddy removed his attention for no longer than might occur during any in-water activity, underlining the danger of all such activities in incompletely controlled epileptics. It is not known whether the buddy was fully aware of the risk which his friend's condition posed: though he knew he was epileptic and took pills he had never seen an attack.

They had one speargun and the victim was holding it when last seen so it is possible he had made an intentional dive to retrieve it if he had accidentally let it slip from his hand, and suffered the fit underwater, but it is unlikely there was time for such a series of events during their brief separation. They were making their way to a more distant buoy when the incident occurred. The medical history is that he suffered only infrequent attacks and these were usually when he was fatigued and seemed to be limited to the early hours of the day, and for this reason he was given permission to play rugby, and swim if accompanied.

EPILEPTIC. 2ND SNORKEL SWIM. MOMENTARY VISUAL SEPARATION FROM BUDDY. EPILEPTIC ATTACK PROBABLE CAUSE OF LOSS OF CONSCIOUSNESS, DROWNING.

Case BH 83/3

Although he (probably) died from a blackout after he hyperventilated to breath-hold dive to retrieve his mask, he had his scuba equipment at hand and could easily have worn it. It is probable that the water clarity misled him in estimating how deep the water was, and his breath-hold diving skill enabled him to put himself at risk. There were 12 experienced divers on a boat dive, the victim and his buddy being among the six set down on a rocky islet as their dive base. He and his buddy dived for 20 minutes then surfaced further than expected from the rocks but were not unduly fatigued by their return swim, and then rested on the rock after removing all their equipment, including their

NEW ZEALAND DIVING-RELATED FATALITIES 1983

CASE	AGE	DIVE SKILL VICTIM	DIVE SKILL BUDDY	DIVE GROUP	DIVE BASE	WATER DEPTH DIVE	WATER DEPTH INCIDENT	WEIGHT BELT ON OR OFF
BH83/1	23	Nil	Not stated	2 Separation	Beach	Not stated	Surface	On
BH83/2	16	Nil	Trained Experienced	2 Separation	Beach	Not stated	Surface	On
BH83/3	29	Experienced	Trained Experienced	Separation Solo	Rock	70'	? ASC ?	On
BH83/4	25	Experienced	Not stated	2 Separation	Beach	12'	Not stated	On
SC83/1	25	Part trained Inexperienced	N/A	Solo	Land	15'	15'	On
SC83/2	32	Trained Experience	Experienced	(9) Buddy	Boat	25'	25'	On
SC83/3	34	Not stated Inexperienced	N/A	Solo	Boat	Not stated	Not stated	On
SC83/4	32	Not stated Inexperienced	Experienced	3 Separation	Beach	15'	15'	Not stated
SC83/5	46	Just trained Inexperienced	Trained Experienced	3 Separation	Boat	85'	? ASC ?	On
SC83/6	30	Both pupils	in a class	2	Boat	30'	Surface	On
SC83/7	22	Trained Experienced	Trained Experienced	2 Separation	Boat	30'	Surface	On

compensators.

Another diver borrowed the victim's mask to recover his and shortly after it was returned a sudden wave threatened to wash all their equipment into the sea. He succeeded in retaining everything except his mask so borrowed his buddy's and made a surface search. He called out that he could see the mask 30 feet below (he estimated) on the sea bed, then he dived. He failed to surface and two minutes later his buddy felt alarmed so attempted a "blind" (maskless) breath-hold dive search for him, but was not successful. Later two scuba divers found the body in 70 fsw water but as there was a strong current it is not certain whether this represents the dive depth. The weight belt did not have a quick-release and it required the efforts of both rescue divers before they could remove it.

SCUBA DIVER MAKING BREATH-HOLD DIVE TO RECOVER MASK. DIFFICULT TO REMOVE WEIGHT BELT AS NO QUICK RELEASE. SOLO DIVE. PROBABLY HAD A POST-HYPERVENTILATION BLACKOUT. BUDDY HINDERED BY LACK OF MASK.

Case BH 83/4

The two divers were spear fishing in 12 feet deep water. After about 20 minutes they separated, and 3-5 minutes later as the buddy was swimming to deposit a fish in their boat he saw the victim lying on the sea bed below, all his equipment in place. He dived and brought him to the surface and got him onto some rocks, ditching the weight belt to make the task easier. He called for assistance and started EAR resuscitation but the victim did not respond. He was known to have had a thoracotomy to remove a bronchiectatic right middle lobe and to suffer from asthma but the most probable critical factor is a post hyperventilation blackout as he had not been known to suffer asthma while at sea or diving, the water conditions were good, and he had shown no signs of any problems when seen a few minutes before he died.

SEPARATION/SOLO. SPEARFISHING. FOUND ON SEA BED. CALM SEA. POSSIBLE POST-HYPERVENTILATION BLACK-OUT. NO BUOYANCY VEST. MEDICAL HISTORY BRONCHIECTASIS AND THORACOTOMY WITH REMOVAL RIGHT MIDDLE LOBE. ASTHMA.

NEW ZEALAND DIVING-RELATED FATALITIES 1983

WEIGHT BELT WEIGHT LB	CONTENTS GAUGE	BUOYANCY VEST	EQUIPMENT TEST	REMAINING AIR	EQUIPMENT OWNER	WET SUIT	COMMENTS
Not stated	N/A	No	N/A	N/A	Loan	Jacket	Weight belt without quick weights release; surface problem; buddy too tired to help; good swimmer.
Not stated	N/A	No	N/A	N/A	Not stated	Yes	Only short separation time. Epileptic.
Not stated	N/A	Off !	N/A	N/A	Own	Yes	Scuba diving; lost mask, so breath-hold dive to find ! ?post-hyperventilation blackout?
Not stated	N/A	No	N/A	N/A	Own	Yes	Medical history of thoracotomy for Bronchiectasis. Asthma. Post-hyperventilation blackout?
Not stated	Not stated	No	Yes	Not stated	Own	Part only	Ex-pub, diving for eels in river. Non-divers nearby.
33	Yes	Not	Yes Inflated	1/2 full	Own	Yes	Separation by sudden water surge; buddy also at risk.
Not stated	Not stated	Not Inflated	Yes	Not stated	Loan	Yes	Friend in boat waited and waited; found 2 days later.
Not stated	Not stated	Not stated	Yes	Nil	Not stated	Yes	Refresher dive with 2 experienced divers; poor visibility, separation.
Not stated	Yes	inflated	Yes	Nil	Own	Yes	Surfaced unconscious: CAGE, Coronary artery atheroma; weight belt as worn could not be released quickly.
20	Yes	Inflated	No	Near full	Own	Yes	1st sea dive: class, surface acute cardiac arrhythmia?
21	Yes	Fault	Yes	Low	Loan	Yes	Inhalation of vomit. Wore torn

Case BH 84/1

The victim, who was wearing swim shorts and booties, was snorkelling with a companion when he suddenly started beating his chest with his fists, taken by his companion to mean that he had heart trouble. The buddy went to his aid but when he reached rocks near the shore the victim collapsed, his breathing becoming irregular. His breathing ceased prior to his being placed in the Land Rover sent to transport him and EAR was commenced before he was moved. Ambulance personnel gave oxygen and applied ECC in an attempt to resuscitate him but he did not respond.

He had a history of a heart complaint, though to what degree this was known to his buddy is unknown. At the autopsy the mitral valve was described as admitting three fingers easily but the cusps were not overtly abnormal; the heart was enlarged, mainly due to dilation with some ventricular hypertrophy; and there was a double right coronary artery arising from the right coronary cusp. There were petechial haemorrhages and ecchymoses present on the external surface of the heart over the track of the coronary vessels, and also the lungs, particularly in the hilar region. The appearance suggested gross congestion. The diagnosis was that he suffered "oedema of the lungs consistent with irregularity of the heart beat, consequent on past rheumatic fever". This indicates that the pathologist was unaware of the true medical history as a consequence of the usual practice of non-contact with the regular medical practitioner of victims with medical conditions.

His medical history is that at age 12 he presented with an episode of rapid supraventricular tachycardia which had required electrical conversion. There was another episode at age 16 which required hospital treatment. He apparently experienced numerous episodes which settled spontaneously and he only went to the hospital if such resolution did not occur. He was advised by the specialist who first made the diagnosis, that he was suffering from the Wolf Parkinson White syndrome, that he should lead a full active normal life. He followed this advice by playing football, and he had been a keen scuba diver for over two years. In fact he had been scuba diving prior to his death and only changed to snorkel after emptying his tank. He had

NEW ZEALAND DIVING-RELATED FATALITIES 1984

CASE	AGE	DIVE SKILL VICTIM	DIVE SKILL BUDDY	DIVE GROUP	DIVE BASE	WATER DEPTH DIVE	WATER DEPTH INCIDENT	WEIGHT BELT ON OR OFF
BH84/1	22	Not stated	Not stated	2	Rocks	Not stated	Surface	Off
BH84/2	16	Not stated	Not trained Some	Group Solo	Beach	Not stated	On land	Off
SC84/1	24	Not trained Inexperienced	Trained Experienced	2	Boat	45'	Surface	Not stated
SC84/2	24	Not trained Experienced	Trained Experienced	2 Solo	Dock	38'	30'	On
SC84/3	34	Not trained	Trained	Separation	Rocks	10'	Not stated	On
SC84/4	20	Not trained 1st Scuba Dive	Not trained 3rd Scuba Dive	Separation Solo	Boat	8'	8'	On
SC84/5	43	Not trained Inexperienced	Part trained Some	Separation Solo	Boat	Not stated	Not stated	On
SC84/6	36	Not trained Inexperienced	Not stated Separation	3	Boat	15'	Surface	On
SC84/7	54	Not stated	Not stated	Separation Solo	Boat	35'	Not stated	On

been collecting mussels and was at the surface when the fatal episode occurred, presumably a severe tachycardia. It is thought that he managed to pull himself onto the rocks unaided before he collapsed but as no inquest was held there is no copy of the buddy's deposition in the case records.

SNORKELLING AFTER SCUBA DIVE. BREATH-HOLD DIVING FOR MUSSELS. ACUTE CHEST PAIN AND CARDIAC DEATH FROM WOLF PARKINSON WHITE SYNDROME, 10 YEAR HISTORY THIS SYNDROME. LED ACTIVE LIFE DESPITE CONDITION.

Case BH 84/2

This boy went to the beach with four friends and spent most of his time spearfishing, though they spent most of the time ashore. It is not known how successful he was, but as he was not wearing a wet suit and the water was cold he must have been tired when he came ashore. He rarely smoked but may have smoked part of a cigarette with his friends before they all started to walk back to their car. He then began to feel ill and had to sit down for a time to rest while a friend ran to get his "Ventolin" inhaler from the car. His friends were used to his lagging behind them so were not alarmed at this time but when he became breathless they asked a person in a nearby house to call an ambulance and a passer by started giving EAR resuscitation. It is not known at what stage he died but he could not be resuscitated on arrival at hospital. He had required hospitalisation, two and a half years before, for a severe asthma episode and since then had strictly followed the prescribed routine meditation with "Ventolin" and "Becotide". The autopsy examination showed the presence of active asthmatic changes (widespread mucus plugging with tenacious viscid mucus in the medium sized bronchioles and the bronchiolar basement membranes were thickened with numerous eosinophils present in the surrounding tissues). This death was due to an acute asthmatic episode.

SEPARATION/SOLO SPEARFISHING. NO WET SUIT. COLD WATER. ASTHMATIC ON REGULAR MEDICATION. EASILY BREATHLESS ON EXERTION (?). "NON SMOKER" BUT FEW PUFFS OF CIGARETTE POST DIVE. FATAL ASTHMA WALKING TO CAR.

SCUBA DIVING CASES

Case SC 83/1

After a reunion which included some beer a group of six people went to a nearby river to catch some eels for a hinaki pot. While the others were to swim the victim intended to use his scuba to search in a deeper (15 feet) area in the river. After he had dressed in his wet suit etc., he entered the deeper area where he seemed to remain. His friends became alarmed when he had still not surfaced one hour later and made an unsuccessful search for him. His body was found later in a shallower area of the river.

NEW ZEALAND DIVING RELATED DEATHS 1984

WEIGHT BELT WEIGHT LB	CONTENTS GAUGE	BUOYANCY VEST	EQUIPMENT TEST	REMAINING AIR	EQUIPMENT OWNER	WET SUIT	COMMENTS
N/A	N/A	No	N/A	N/A	Own	No	Wolf Parkinson White Syndrome. Sudden chest pain: cardiac.
N/A	N/A	No	N/A	N/A	Own	Yes	Asthmatic. Fatigue. Cold. Rapid post-dive death.
Not stated	Not stated	Yes Inflated	No	Not stated	Loan	Yes	Third scuba dive. Asthmatic. Pre-dive use of inhaler. CAGE. Difficult to get into boat.
24	Yes	Yes Not inflated	Yes	Low	Own	Yes	Cleaning the hull of ship in dock. Found unconscious. Cause unknown.
Not stated	Yes	No	Yes	Nil	Own	Yes	Rough sea. Solo. Failed to drop weight belt. Crayfishing.
14	Yes	Yes Not Inflated	Yes	Low	Hire Loan	Yes	First use of scuba. Asthmatic. Rough sea; cold; lost mask; failed drop weights; inexperienced buddy separation; solo swim.
Not stated	Yes	Yes Not Inflated	Yes	Nil	Own	Yes	Failed to ditch weights. Alcohol was a health risk factor. Started dive low on air.
Not stated	Yes	No	Yes	1/3	Loan	Yes	Asthmatic. First use of scuba. Surfaced solo. Crayfishing.
Not stated	Yes	Yes Not inflated	No	Half	Own	Yes	Surfaced in distress then died. Coronary artery disease. Possible air embolism.

Although he was proud of his scuba knowledge it was found that he had not completed his training and was uncertificated. His equipment was checked and no defect noted but there is no record of any check to note the remaining air pressure.

SOLO. INCOMPLETELY TRAINED. INEXPERIENCE. FAILED TO DROP WEIGHT BELT. NO BUOYANCY VEST. RIVER DIVE? ALCOHOL?

Case SC 83/2

The charter boat brought a party of nine experienced divers to an offshore islet dive site and anchored in 25 fsw deep water 50-60 feet from the shore. The divers entered the water as three pairs and a threesome, the victim and his buddy being last to enter. They planned that the buddy would catch crayfish while the victim was to carry the catchbag. Five of the divers surfaced and had been collected, then the skipper saw the buddy surface and start to signal for help, obviously in distress. After he had been brought aboard he described how a sudden surge had tossed him to the surface and torn off his fins, and told of seeing the victim rushed past him by the surge. An immediate surface search was not successful and the skipper then organised an underwater search by the scuba divers. This also was unsuccessful until the catch bag was located near the white water line and outside the original search area. The skipper and another diver now searched this area and located the victim's body, to which they attached a line and thereby were able to pull it up. He was wearing a 5mm wet suit and his 33 lbs weight belt was considered acceptable though a few pounds more than usual. He would have found himself considerably overweighted in the white water area where the bubble content of the water reduces its density. During the recovery of the body the buoyancy vest was inadvertently activated and inflated, which demonstrated that it was in good condition. It is possible that the victim lost his regulator and was unable to recover it while he was tumbled about in the rushing water. The autopsy reported the cause of death as drowning with terminal inhalation of some gastric contents. No signs of any head injury were found.

WATER POWER. SUDDEN POWERFUL UNEXPECTED SURGE AMONG ROCKS AND WHITE WATER. DIVE LOCATION DANGER NOT RECOGNISED. BUDDY EQUALLY AT RISK. EFFICIENT RESPONSE TO EMERGENCY BY SKIPPER OF DIVE BOAT.

Case SC 83/3

During an outing with some friends and relatives the victim intended diving for scallops. He was rowed out in a dinghy about 500 yards from the shore, with three non-divers remaining in the 8 foot boat when he dived. He soon surfaced and reported that there were scallops there but they were too small, so he held onto the stern of the dinghy and they towed him a bit further out from the beach. He asked that the boat follow his bubbles, then dived again, but because of

the chop and the wind his friends soon lost track of the bubbles. The boat remained in the dive area for one hour before any real anxiety was felt. One of the people in the boat twice thought he had seen something at the surface but these sightings were false, nothing being found when the boat reached the areas of sightings. They returned to the beach after making an unsuccessful surface search and waited there for a time before notifying the police. Shore line and diver searches that day and the next were unsuccessful but a diver found the victim's body two days later, all equipment in place. There was no direct comment to confirm that a weight belt was worn and still in position.

The equipment was reported to be in good condition, though no record was made of whether there was any remaining air. He wore a compensator which could only be inflated orally so was of no value in an underwater emergency situation. The scuba had been borrowed but there is no indication of its source. As he was apparently untrained and had very little diving experience, if his friend is correct, the loan of equipment to him was irresponsible. When he was found he held his regulator in one hand and the catch bag containing scallops in the other, indicating that he had not realised in time his critical need to surface, had concentrated on retaining his catch rather than on his own survival. There is no record of whether he wore, retained, or ditched a weight belt.

UNTRAINED. INEXPERIENCED. SOLO DIVING FOR SCALLOPS. BUOYANCY VEST OF ORAL INFLATION TYPE. WEIGHT BELT NOT STATED. REMAINING AIR NOT STATED. NO CONTENTS GAUGE OR RESERVE SYSTEM. FAILED TO DROP CATCH BAG. BORROWED SCUBA.

Case SC 83/4

The purpose of this dive was to refamiliarise the victim to scuba as he had not dived for about three years: nothing is known of his previous experience or whether he was trained, and the source of his equipment (his own or borrowed or hired?) is unknown. For this reason his two friends were swimming in a leisurely way in water no deeper than 25 feet. They swam in line, the victim as the tail ender. When they noticed his absence they were in water of poor visibility, 15 feet deep. One immediately surfaced to look for his bubbles and the other buddy started to make an underwater search. They were now concerned for their friend's safety so they started a search pattern dive in the area where they had last seen him and soon found him face down on the sea bed. As soon as they surfaced him they started giving EAR and continued this as they towed him to the beach. There was no response to continued resuscitation efforts and the police were notified.

The equipment was checked for the police by a diver with extensive experience and found to work correctly. The tank was empty when examined. There is no comment concerning whether a weight belt was worn and retained/dropped.

The autopsy was conducted with full awareness of the special relevance of diving factors. It was concluded that death was due to drowning, with inhalation of vomit as a terminal event. It is assumed that he must have either failed to recognise his low air situation or have attempted to swim to contact his two friends to tell them his air status, then found himself out of air and failed to try to reach the surface, so drowned. A possibility exists that he made an emergency ascent and suffered a cerebral arterial gas embolism without there being signs of any pulmonary barotrauma, lost consciousness, sank, and drowned. This is unlikely.

TRAINING NOT STATED. EXPERIENCE NOT STATED. OUT OF PRACTICE. THREESOME OF DIVERS. CALM SHALLOW WATER. SEPARATION WHEN LAST IN LINE. NO CONTENTS GAUGE OR RESERVE. NO BUOYANCY VEST. PROBABLY FAILED TO DITCH WEIGHT BELT. PROBABLY OUT-OF-AIR SITUATION.

Case SC 83/5

Members of several dive clubs were together on this charter boat trip to hunt for scallops. The boat carried fifteen persons, ten of whom were divers. There was no formal organisation and each club was expected to assess its own divers' fitness for the 85 fsw dive. The victim had only recently completed his basic scuba course and had made three sea dives on the course and four subsequently, two on each of two days a fortnight after completing the course. The fatal dive took place ten weeks later.

The president of the "host" club was an instructor and was aware of the inexperience of some of those aboard though he did not formally vet any of them as he believed this to be the responsibility of their own clubs. He was first in the water, with his buddy, when they reached the scallop bed, and had got back onto the boat only a short time before the victim came to the surface and gave a distress signal. His first intimation that anything was wrong was when the boat pulled up its anchor and started to move to reach the victim.

One of the divers aboard was involved in a project which involved tagging scallops, a Government investigation, and he noted the absence of any safety officer, or indeed of any control of the divers, so asked a non-diver who was remaining on the boat to make a log of divers as they entered and left the water. This information was produced by the police but no formal deposition of the facts by the witness was brought forward at the inquest.

The victim dived with two others, fellow members of his diving club. They surfaced after diving for 20 minutes, quite untroubled by the absence of the victim, assuming he had decided to surface without them. The scallop bed was at 85-90 fsw and a fairly level area. The victim's depth gauge had a maximum depth recorder which showed he had been at 90 fsw. Some of the divers declined to dive at this spot, deeming it too deep. The instructor gave it as his opinion that it is every diver's responsibility to decide whether or not to dive. This may not hold true where a commercial rather than an informal social dive situation exists.

It is probable, though not stated, that the victim's buoyancy vest was inflated when he surfaced. It is known that its carbon dioxide cylinder had been fired and that his tank, which had a contents gauge, was empty. The equipment was tested and was found to work correctly. His dive had lasted about 17 minutes.

The victim was described as surfacing only a short distance from the dive boat (launch). He raised his arm in the distress signal and did not respond to a call. As he was reached by the boat he rolled onto his back, let out a gurgle, his pupils became dilated, and froth appeared at his mouth. His weight belt was undone and he was brought aboard. No pulse or breathing was found so full CPR was commenced, which produced a faint pulse for a time. The instructor, who had entered the water as soon as it was apparent something

serious had occurred, noted that his weight belt buckle was twisted and had slipped towards the left side so was difficult to release: it was an unusual double-buckle type of belt. The evidence points to him having failed to remain near to his buddies and to have been too engrossed in scallop hunting to watch his contents gauge. In his inexperience he failed to note the warning his regulator would give of the development of a low air situation so would be suddenly faced with a no-air situation at 90 fsw depth, alone. He would probably be overweighted (nobody offered advice and he probably never dived any significant depth previously) and his weight belt would be difficult to drop even in a non-panic situation, so the buoyancy vest would be used to initiate his "emergency lift-off". Unfortunately this ascent was not associated with adequate venting of air from his lungs.

The autopsy was conducted with the special care any death like this requires, with X-ray before commencing and search for evidence of air embolism during the examination. Fractured ribs, from resuscitation efforts, were noted plus air shadows over the heart outline and air in the right ventricle, left pulmonary artery, the aortic arch, in the axillae and anterior neck, and some in the right mid zone indicating a possible area of the lung rupture. This massive air entry would defeat any resuscitative efforts.

NEWLY TRAINED. GROSS INEXPERIENCE. GROUP OF THREE. EXCESSIVE DEPTH FOR EXPERIENCE. NO DIVE DISCIPLINE. SEPARATION/SOLO DIVE. "BUDDIES" IGNORED ABSENCE OF VICTIM. SENIOR DIVER DID NOT ACT AS DIVE MASTER ON BOAT. BUOYANCY VEST INFLATED. FAILED TO DROP WEIGHT BELT. WEIGHT BELT TWISTED AND DIFFICULT TO REMOVE. HAD CONTENTS GAUGE BUT OUT OF AIR. MASSIVE AIR EMBOLISM. OVER-VIGOROUS ECC DAMAGES RIBS.

Case SC 83/6

The seven trainee divers were allowed to accompany a club boat dive in order to take their sea tests. They all checked their own and their buddy's equipment before entering the sea, the instructor being present and watching. The victim, who wore a wet suit, compensator, and 20 lb weight belt, had a contents gauge and a full tank and was in the third buddy group to start the surface swim using snorkel. The test was planned to be a surface swim in full equipment to a rock and back, about 400m, followed by a scuba dive. After swimming about half way to the rock the victim pulled on his buddy's arm and indicated he had some trouble, so the buddy orally inflated his vest for him and called to another diver for assistance. The victim appeared to be breathless so they started to tow him back to the dive boat. Their actions were seen by the instructor, who was following the fourth pair, and he soon reached them and took command, giving in water EAR as the other two towed the victim to the boat. He was so buoyant there was no need to ditch his weight belt. There was no apparent response to the EAR and after being brought aboard the boat he was not breathing, no pulse was apparent, and he was unconscious, so CPR was instituted and this was continued by various divers during their return to land on a boat which came in response to their radio call for assistance. Supposedly their request for the helicopter medical response team was discounted because someone in the control chain of command stated "a heart attack doesn't warrant a helicopter", a fact not noted at the inquest. This rescue service had only very recently become operational and the person responsible may have made a judgement appropriate to a land incident, forgetting that the present circumstances were different. This may not have made any difference to the outcome, however. The victim was not known to have any ill health and had a physically demanding job.

The victim's heart was noted to start beating and he began to breath as the boat was docking. An ambulance awaited their arrival and emergency therapy was given during the journey to the hospital, which lasted half an hour. His condition changed when they were 10 minutes from the hospital and CPR was resumed. Death was certified 40 minutes after he arrived at the hospital.

The instructor noted two valuable lessons from this tragedy, the first being the importance of maintaining effective buddy contact with frequent checking even when at the surface, and the second was the need to check the emergency medical supplies before every dive trip as the oxygen bottle was empty when needed. The alertness of the skipper was recorded with appreciation as he pulled on his anchor line when he saw what was happening, thereby drawing the boat nearer to the returning divers, to their benefit.

At the autopsy there was marked haemorrhagic oedema of the lungs with only tiny areas of aeration. Multiple fractured ribs were noted, the result of enthusiastic resuscitative efforts. The pathologist concluded that death was from heart failure, which he thought resulted from a sudden cardiac arrhythmia triggered by an inhalation of salt water.

IN TRAINING. SURFACE SNORKEL SWIM IN FULL EQUIPMENT WITH BUDDY. BECAME BREATHLESS. BUOYANCY VEST ORAL INFLATION TYPE INFLATED ON REQUEST OF VICTIM BY BUDDY. GOOD BUOYANCY ACHIEVED. EXCELLENT HELP FROM BUDDY, OTHERS, INSTRUCTOR. EAR RESUSCITATION COMMENCED IN-WATER RADIO ASSISTANCE CALL PROBLEM. EMPTY EMERGENCY OXYGEN CYLINDER. ACUTE CARDIAC ARRHYTHMIA POSSIBLE CAUSE OF DEATH. OVER-VIGOROUS CPR FRACTURES RIBS. IMPORTANCE TRUE BUDDY DIVING "EVEN AT THE SURFACE".

Case SC 83/7

Three friends decided to go diving for crayfish from the 8 foot runabout owned by one of them. The victim had made 14 dives since his scuba training 2 years previously and was using borrowed equipment both he and his buddy checked before he used it. He removed 3 lbs from the belt after this check. One diver descended and checked the anchor, then boarded the boat again and remained there as boatman and surface cover when the other two made their dive. They had an uneventful dive for 45 minutes, maximum depth being 30 fsw, then surfaced together. The buddy checked and established that while the victim had 400 psi remaining air he had 650 psi, so agreed to his suggestion that he swim back to the boat with the catch bag and its single crayfish while the buddy made a final search for more cray fish. The sea was moderate with a northerly wind, not uncomfortable conditions for diving, and the boat only 30-50 yards distant. Shortly after he dived he heard the boat's engine start then stop again so he surfaced to see what was happening. It is not known whether the victim was feeling fatigued by his dive as he started his return swim.

The diver who remained in the boat saw the victim at the surface about 50 yards from him and about 10 yards from the shore line rocks when he raised one hand above his head and called out, indicating a desire to be picked up. He seemed to be looking around for

his buddy, so when he raised his hand a second time the witness thought something was wrong with the buddy as the victim did not appear in any difficulty. The witness started the boat and motored over to him after an initial difficulty in getting the anchor free. When first sighted he had been floating well above the swell and no waves were breaking over him, then he was seen swimming towards the rocks, but he had drifted into a gut among white water by the time the boat reached him. He was face down, his snorkel above the water, and it took a few moments for the witness to realise that he was not moving, then he threw the anchor overboard, jumped into the water, and swam to give him assistance. On reaching the victim the witness, the third diver of the party, turned him face upwards and noticed that his mask was full of water and vomit so pulled it off. He then noticed a white froth in his nose and mouth and his facial cyanosis. With difficulty he dragged him onto the rocks and tried to give him a few breaths by EAR but waves were breaking over them so he ditched the victim's weight belt and scuba and pulled him out of the water. Shortly after this he was joined by the buddy, who had first returned to the boat but discovered it was unoccupied and then heard him calling. Together they applied CPR but obtained no response.

The autopsy revealed evidence that inhaled food had reached the smaller bronchi and the cause of death was given as drowning plus inhalation of vomit. The reason for his initial problem is unknown but the equipment check revealed two adverse factors, a tear in the back-pack-attached compensator (unknown brand, tear pre dated the incident) and a leak past the mouthpiece which could cause some salt water inhalation. The snorkel he was using belonged to his buddy, who gave it to him for his return swim as he was not carrying one.

TRAINED. MODERATE EXPERIENCE. BORROWED EQUIPMENT. TORN BUOYANCY VEST NOT NOTED AT PRE-DIVE CHECK BY TWO DIVERS. DID NOT CARRY SNORKEL, BORROWED SNORKEL FOR SURFACE SOLO SWIM. LOW AIR/OUT-OF-AIR STATUS. BUDDY MADE SOLO LOW-AIR DIVE LEAVING COMPANION AT SURFACE. VALIANT ASSISTANCE OFFERED BY BOTH COMPANIONS. DIFFICULTY EXITING IN WHITE WATER ONTO ROCKS. INHALED VOMIT.

Case SC 84/1

The victim had been a snorkeller for 4 or 5 years and was keen to try scuba diving. His friend had taken a scuba course nearly three years previously and apparently offered to give him some instructions, at the same time taking him collecting kina. The victim was a severe asthmatic who used one "Ventolin" inhaler per month, his allergy being to pollens.

This was the third time he had used scuba, the second being the previous day in the same area, to 20 fsw. He was using borrowed equipment and was with two companions, diving from a 14ft outboard runabout. They had difficulty getting the anchor to hold where they had dived the previous day so moved closer in to the island where the water was expected to be shallower. Before they entered the water the buddy told the victim to breath out as he ascended, to breath shallowly if he needed a breath during ascent, and to travel at the speed of his bubbles, and showed him the basic hand signals. The victim told his buddy that he had a tendency to hold his breath during ascent. Instruction completed, they dived.

Before he entered the water the victim had used his Ventolin inhaler, then he tucked it into one sleeve of his wetsuit and entered the water. While in the water at the side of the boat he was out of sight of his buddy, who entered the water from the other side of the boat (but the man left in the boat saw him use the inhaler a further three times). He replied to his buddy that he was all right when asked how he felt before they descended the anchor line. Water depth was about 45 fsw and they each carried a sack for the kina they collected. Visibility was about 15 feet and they kept in visual contact at all times, exchanging signals when they had full sacks and then both started to ascend. Although the victim started a little before his buddy the latter overtook him and surfaced first, about 30 feet from the boat and 7 feet distant from him. He again answered a query from his buddy, saying he was all right, then suddenly one of his legs stiffened and he let out a cry of pain, so his buddy told him to hand over his sack of kina and this he did without speaking. His compensator was noted to be inflated at this time. The buddy now signalled to the man in the boat to collect the victim first, and told the victim to lie on his back and ignore the tide current as the boat was coming to collect him. At this time he gave another cry as if he had pain, but did not indicate the nature of his problem, and lifted the face mask from his face. He then slowly drifted away from where the buddy was floating holding both bags of kina. Gradually the buddy tired and before the boat returned for him he had emptied one bag to lighten his load.

The man in the boat saw them surface, one a little in advance of the other, then give the arranged signal requesting him to collect them. They had been diving about 15 minutes. When he reached the victim he was floating on his back with a vacant look on his face and did not respond to speech or when thrown a line with an inflated tube attached. He realised something serious had occurred and managed to tie a line to one of the victim's arms. It was very difficult but he managed to get him in the boat by securing a line to his backpack belt and pulling, first ditching his weightbelt. He was still breathing at this time but was unresponsive to questioning. The rescuer gave him a couple of breaths by EAR then collected the buddy (who found it difficult to board the boat).

The buddy started giving EAR but soon tired so asked the boat man to replace him. During their return trip they passed some fishermen who had a radio and they sent a message to alert the shore emergency services of a seriously ill diver. Both his friends continued their CPR efforts on arriving at land until the ambulance arrived. It is assumed from the autopsy finding that he was intubated, but there is no information on whether this was in the ambulance or at a hospital, nor is it clear whether he was still living when he reached land. At the autopsy both lungs were over-inflated and somewhat waterlogged "but there was no evidence of mucous plugging". The microscopy of the lung tissue samples was reported as "the small airways showed quite marked residual mucous plugging. Elsewhere the lungs show congestion, some intra-alveolar haemorrhage, and oedema. Although these appearances are complicated by resuscitation, it is clear that there was a significant degree of small airways obstruction consistent with asthma at the time of death". The official finding as to the cause of death was "Severe Bronchial Asthma in the course of Scuba diving". On that basis of the history it is highly probable that the critical factor was a Cerebral Arterial Gas Embolism (CAGE), symptoms beginning a short time after he surfaced. He was totally untrained and uninformed, he had active asthma symptoms before he made his descent, and was aware himself of a tendency to breathhold during ascent. It is unfortunate the Coroner made no comments on the gross impropriety of lending such a man scuba or taking him

on a scuba dive. It may be thought the buddy's course failed to make him understand the basic facts of safe diving as he saw nothing wrong in his actions.

The Coroner did not consider it necessary for there to be any inspection of the equipment used by the victim.

UNTRAINED. GROSS INEXPERIENCE. THIRD SCUBA USE. COLLECTING KINA 45 FSW. APPARENTLY CORRECT RATE ASCENT BUT NOT WITH BUDDY CLOSE TO HIM. INFLATED BUOYANCY VEST. CRIED OUT THEN UNRESPONSIVE AT THE SURFACE. BORROWED EQUIPMENT. ACTIVE ASTHMA WHEN DIVED. AIR EMBOLISM. (CAGE).

Case SC 84/2

A ship heavily fouled by marine growth was requiring hull cleaning while loading timber in harbour and a small firm of diving contractors arranged to perform the job. This was a normal type of contract for them. There were four divers involved though only two were in the water at any one time while one was standby on the wharf. They used scuba, each dive usually lasting for 60-70 minutes, the diver then surfacing to refill his tank while an other diver took his place. Some of the growths were too heavy for the scrubber brush to clear so one of the divers was using a spade to clear such areas. The divers worked independently of each other.

The victim was not diving the first day because he was recovering from a mild "flu" infection and was undertaking a non-diving job, but the second day he declared he was recovered and fully fit to dive. He was experienced, having been scuba diving 10 years, but untrained, and he had been undertaking commercial diving jobs such as this for 2 years. The ship had a somewhat flat hull bottom with bilge keels as deep as the main keel and deeper than the main area of the bottom. The maximum draft unladen was 8.78 m, and when loaded 9.39 m. The harbour depth here was about 11.4 m.

First intimation of trouble was when it was noticed that the victim had not surfaced as expected and no bubbles could be seen breaking at the surface. They hurriedly made a search but could not find him beneath the ship so informed the police, asked for other divers to be sent to assist the search, and themselves organised a painstaking search, the three divers swimming from bow to stern with a rope between them from port to starboard to keep in contact. It was during one of these searches that they found the spade used by the victim, then one diver put his hand down and felt the victim, face down on the harbour floor. The finder guided another diver's hand to confirm the discovery and he reacted by a sudden ascent holding the body, the safety rope between the search divers pulling the finder unceremoniously up with him. The victim's vest was inflated and his tank contents gauge read 500 psi, though it was a faulty gauge and the actual remaining air was about 400 psi.

Check of the equipment also showed that the pressure in the hose proximal to the regulator was low, making it harder to obtain air than it should have been. Neither finding explains the incident.

At the autopsy there were several unexplained deep puncture marks under the jaw and there was marked mucosal damage of the buccal mucosa of both upper and lower lips, mucosal loss of the insides of the cheeks and lacerations of the tongue. Findings such as this suggest an epileptiform fit occurred (reason unknown). There was no pneumothorax but both ventricles contained frothy blood and there were air bubbles in the surface vessels over both cerebral hemispheres and filling the basilar artery. Both lungs were over-inflated and waterlogged but fluid could not easily be expressed. There was frothy fluid in the main bronchi, larynx, and trachea. It is difficult to account for an air embolism occurring as he was not out of air, was still wearing his 24 lb weight belt, had an uninflated buoyancy vest, and was beneath the hull where it would be unlikely a body would reach if it sank after some cerebral air embolism caused loss of consciousness during a hurried ascent. No explanation has been found for the punctures beneath the jaw. The autopsy did not reveal any signs of unresolved respiratory tract infection, though the pathologist was looking for such evidence.

During the loading a log fell into the water and this might have created a water movement which tossed him about, though no such effect was noted by the other diver. No scenario has been put forward which can explain the known facts.

UNTRAINED. EXPERIENCED. COMMERCIAL CLEANING HULL OF SHIP LOADING AT WHARF. TWO DIVERS WORKING SOLO BENEATH HULL. NO SAFETY LINES. WEIGHT BELT DITCHED. BUOYANCY VEST NOT INFLATED. SOME REMAINING AIR. IMPERFECT REGULATOR FUNCTION. IMPERFECT CONTENTS GAUGE. MYSTERY OF PUNCTURES JAW SKIN. APPARENT AIR EMBOLISM. CAGE. LOG FELL INTO WATER.

Case SC 84/3

Because of the swell it was decided to dive from the shore rather than from their aluminium run about, so they dropped off their gear on the rock point they intended using as their dive platform, leaving their non-diving friend to watch its safety while they tied up the boat at the wharf about 200 yards away. After they had walked back and kitted up they walked through a gap in the rocks to the seaward side of the point, decided the sea there was too rough for them, so returned to the wharf side of the point and entered the water. Their dive purpose was to collect crayfish.

Within a matter of minutes they became separated but they continued diving separately. The victim was seen to surface a number of times in a normal manner and it was only after his buddy surfaced an hour later with an empty tank that any anxiety was felt, as the victim was "heavy on his air" and always emptied his tank first. The buddy had been diving in water depths between 10 and 50 feet, as was his habit, working in from the point towards the wharf and expected his friend to have a similar plan. When he had last surfaced, 5 minutes before the buddy came ashore, he had been about 30 feet out and had been seen to clear his mask and to snorkel at the surface for a short time before again going down.

His buddy climbed the rocks to get a better view of the water but could see no sign of the missing diver so they took their boat and made a surface search of both sides of the point, then informed to police. The chop was now too dangerous for their runabout but the police launch and two other boats continued the search till dark while two Coast Guard divers made an underwater check. Next day searching resumed using two boats, an inflatable, and five divers. The victim was found 20-30 feet from the point, in 10 feet deep water, after searching for an hour. His equipment was complete and his mask, a little displaced, contained some blood stained water. Visibility, which was 20 feet the previous day, was now reduced to 4 feet. The buddy was certified but the victim was not and had picked up his knowledge by diving with the buddy over

ten years. He was said to be a common-sense diver not likely to take risks, one who had never previously had any trouble while diving. The autopsy showed evidence of drowning. When checked his tank was empty but the equipment was in good order. The weight of the belt was not recorded nor whether its quick-release worked: it is not known why he did not drop his weight belt or cry for help, but the lack of a buoyancy aid plus diving habit of continuing to dive till completely out of air critically compromised his safety.

UNTRAINED. EXPERIENCED. SEPARATION/SOLO. DIVING FOR CRAYFISH. SHALLOW, MODERATELY ROUGH WATER NEAR ROCKS. LOW-AIR OR OUT-OF-AIR SITUATION. HAD CONTENTS GAUGE. NO BUOYANCY VEST. FAILED TO DITCH WEIGHT BELT.

Case SC 84/4

Against the wishes of his parents this young man, who was totally ignorant of scuba diving and suffered from asthma (he was on regular, four times daily, "Ventolin" and "Becotide" aerosol inhalations plus "Nuelin" 250mg tablets twice daily) had decided to go diving with a friend. They hired scuba equipment, signing a declaration of competence in diving. The friend was also totally untrained and was making his third scuba dive, while this was the first time the victim had used scuba. He was wearing a wet suit, mask and flippers belonging to the buddy, and a hired compensator and weight belt as well as hired scuba unit.

They swam out from a beach after an initial problem with the victim's regulator, but this functioned well after using the purge button. There was a 10-15 knot wind and visibility was only 3 metres. They snorkelled out parallel to the beach, then the victim said he felt cold and sat on a rock while his buddy dived for paua. He borrowed the victim's weight belt because he was too buoyant with only his own but he returned it before they started a swim back towards their entry point. The buddy remained at the surface, his buoyancy vest part inflated, while the victim swan a little ahead and underwater. The buddy became fatigued and unable to keep up with the victim so diverted to the beach, the victim continuing to swim parallel to and 20-30 metres from the beach.

Just after the buddy care ashore he saw the victim surface, maskless and shouting for help. He submerged, resurfaced and cried out again, then submerged and failed to reappear. The buddy removed his tank with the assistance of a nearby fisherman (who then had the foresight to send a message to the police) and swam out to where he had last seen his friend. He was too buoyant however to be able to descend to search for him and unable to remove his wet suit in the water to reduce his buoyancy, so returned to the beach. The body was found after an intense search involving beach, boat and helicopter searches. The body was found floating face down one metre below the surface in 2-3 metre deep water, all equipment in place: the weight belt was on and the compensator was uninflated (oral inflation type). Remaining air was 300 psi. The autopsy finding was death due to drowning without evidence of active asthma. Both divers had a little snorkelling experience.

UNTRAINED. GROSS INEXPERIENCE. FIRST USE SCUBA. BUDDY'S 3RD USE SCUBA. HIRED AND BORROWED EQUIPMENT. SEPARATION/SOLO. COLD. BUDDY FATIGUED. SURFACED WITHOUT MASK AND CALLED FOR HELP. BUDDY TOO BUOYANT TO DIVE TO SEARCH. UNINFLATED ORAL INFLATION TYPE BUOYANCY VEST. ASTHMATIC ON REGULAR TREATMENT BUT THIS NOT CRITICAL FACTOR. FAILED TO DITCH HIS WEIGHT BELT.

Case SC 84/5

The 15 year old son of the victim described the dive events clearly. He and his eight year old brother accompanied their father on a friend's boat to some islands to collect crayfish and scallops. The two adults were trained and his father, after being away from diving for some years, had been diving regularly for the past 12 months. He was not trained, "though studying for the Basic Scuba Diving Certificate". They made dives at three locations. At the first the victim and his son dived together, the latter having 1000 psi remaining from an initial 3300 psi while his father used 3000 psi. At the second island the two men dived, the victim using a fresh tank and having 1400 psi remaining on surfacing. They had lunch after moving the boat to a little bay and then the friend and the boy descended the anchor line and collected scallops from the adjacent sea bed. When the boy was down to 500 psi they surfaced and saw the victim swimming at the surface towards the boat. When they reached the boat he had again dived. They estimated that his tank would then probably contain 500 psi air. The boy used up his air swimming near the boat, boarding it when his tank was empty. A short time later his buddy returned and also boarded the boat. It was some time before they realised the victim had not returned and they could see no bubbles. They rowed around the area looking for the missing diver but found nothing so notified the police.

The body was found floating out at sea about eleven weeks later, all equipment in place. The equipment was tested and no faults discovered, though his buoyancy vest was damaged during recovery of the body. Identification was through dental records. The autopsy was of limited assistance because of the extensive decomposition. He had a history of excessive alcohol intake and smoked 25 cigarettes a day but had no shortness of breath. He was regarded by his son as "not totally fit but fit enough for diving".

TRAINED. EXPERIENCED. CRAYFISH/SCALLOP HUNTING. SEPARATED/SOLO DIVE. STARTED DIVE LOW ON AIR. IMPERFECT BUDDY SYSTEM. BUDDY/ PART TRAINED BOY. HAD CONTENTS GAUGE AND BUOYANCY VEST. FAILED TO DITCH WEIGHTS. IMPERFECT HEALTH FROM ALCOHOL. DELAYED RECOVERY OF BODY.

Case SC 84/6

Two divers decided to take a friend with them diving for crayfish. The wife of one of them remained in the boat, which was anchored about 100 metres from the shore in shallow water. It was the first time the victim had used scuba and they loaned him the necessary equipment. Water depth was 10 to 15 feet, visibility was 20-30 feet, and there was some kelp on the rocky bottom. While one kept close by the victim at all times underwater the other diver seems to have gone his own separate way. After a time the two divers surfaced and a little later the third one joined them. They checked the victim's contents gauge, which showed between 800 and 1000 psi: they had larger tanks and over 1000 psi remaining so when he said he wished to return to the boat they decided to swim underwater while he swam at the surface. They saw he was making good progress, then they submerged. They had surfaced 30-50 metres from their boat and he probably covered little of this distance before he lifted his head, took off his mask, and cried out "HELP!" then disappeared from view.

The person remaining in the boat was unable to offer him assistance and it was about 3 minutes before the two divers surfaced and learned what had occurred.

They dived and soon found the victim on the sea bed, unconscious. He was brought up and EAR was performed in the boat but he did not respond. His tank still contained 700 psi air when tested so it is uncertain whether his return swim was started using snorkel or regulator. It is not on record whether he wore/ ditched/retained a weight belt, but almost certainly he wore and did not ditch a weight belt.

The equipment was tested and no faults noted. There is no mention of a buoyancy vest being worn. The autopsy showed only the changes of drowning. There is no report of lung sections being examined for evidence of asthma but there is nothing in the accounts of the incident to implicate any other factors than the victim's gross inexperience and the solo situation resulted from the surface separation from his friends.

As a footnote it is worth recording the remark of one of the divers who loaned him the equipment well aware that he had never before used scuba: "Had I been aware that he was asthmatic there is no way I would have let him dive". A strange evaluation of the relative risks of Asthma and Total Ignorance of Scuba use.

UNTRAINED. GROSS INEXPERIENCE. FIRST SCUBA DIVE. SEPARATION/SOLO SWIM AT SURFACE. SURFACE PROBLEM. REMOVED MASK AND SANK. HAD AIR REMAINING. HAD CONTENTS GAUGE. .NO BUOYANCY VEST. BORROWED SCUBA EQUIPMENT. CALM SEA. ASTHMATIC BUT THIS NOT RELEVANT.

Case SC 84/7

There is no record of the training or experience of either the victim or his buddy (his son). They and two non-diving friends went in the victim's boat to an island and set out a long line for fish, then journeyed on to another bay where they planned to dive for crayfish off the rocky shore. They entered the water, here 35 feet deep, and descended to the bottom together but in the poor visibility, 15 feet at best, became separated. Following their usual practice when separation occurred, the buddy surfaced to wait for his father to rejoin him. The people in the boat indicated to him that the breaking bubbles showed his father was continuing on to wards the rocks so he resubmerged and attempted to find him, but was unsuccessful so again surfaced. He now attempted to reach his father by swimming at the surface to the area of the bubbles but this proved difficult because the surface chop slowed his progress and he could not gain on him. Then the victim broke the surface 40-50 feet from him and appeared to be in a distressed state. The buddy immediately swam as fast as hi could to him, reaching him shortly after he ceased struggling and had started to sink.

The buddy called out to those in the boat to come as quickly as they could, then "pumped up" the compensator his father was wearing and his own, ditched his father's weight belt, and gave him support until the boat reached them and got them aboard. They applied EAR during the return to land. Fortunately they passed a launch which had a radio and sent a message ahead of them to have an ambulance waiting their arrival, and a man from the launch came and took over the job of driving their boat, a vital piece of help as the person in the group not able to give EAR was unskilled in managing a boat.

The autopsy was preceded by a chest X-ray, which did not show any pneumothorax, and the pathologist then proceeded with an examination directed at establishing whether there was any air in the major blood vessels, and found none. He commented that "the appearances of the lungs were not suggestive of barotrauma". When the coronary arteries were examined it was noted that there was a 70% stenosis of the proximal left anterior descending artery with a focus of severe narrowing due to atherosclerosis involving the origin of a diagonal branch and severe narrowing from this cause effected the proximal portions of the intermediate and circumflex arteries. There were only scattered changes in the right coronary artery and none were severe. No evidence of any recent myocardial damage was found on microscopy". It was not possible to completely exclude the possibility of a cerebral arterial gas embolism or to assess the relevance of the severe coronary artery disease", said the pathologist, then recorded his conclusion that "in his opinion the appearances are consistent with drowning", and this was given as the official cause of death. Nothing is known of the victim's previous health history.

His equipment was sent for testing and the remaining air was noted as 1500 psi and the maximum depth indicator showed at 130 fsw. It is not known whether this represented the depth of his first dive or related to a previous occasion as no record was made of the dive details in the depositions. The air was clean on test but there is no record of the equipment being tested. It can be assumed from the buddy's evidence that the buoyancy vest worn by the victim could only be orally inflated. The type of vest was not stated so it is not possible to know whether the tank inflation hose was not connected by error or because the vest could not accept such a hose supply.

The reason for this fatality is unknown but it might have been the consequence of an anginal episode which led him to make an emergency ascent which resulted in an air embolism, or the cardiac condition may have led to a rapid cardiac death. Drowning would be the terminal event with either scenario.

TRAINING NOT STATED. EXPERIENCE NOT STATED. SEPARATION/SOLO. BUDDY ACTIVE ATTEMPT TO REJOIN COMPANION. SURFACED IN DISTRESS. ADEQUATE REMAINING AIR. BUDDY MADE CORRECT PERSISTENT ATTEMPTS TO LOCATE AND REJOIN FRIEND. SURFACED IN DISTRESS. BUDDY ASSISTANCE RAPID TO INFLATE BUOYANCY VEST (ORALLY) AND DITCH WEIGHT BELT. CAREFUL AUTOPSY PROCEDURE. CAGE? CORONARY ARTERY DISEASE. POSSIBLE AIR EMBOLISM.

DISCUSSION

Examination of the details of these cases reveals an over representation of several factors commonly regarded as being highly adverse to safety. The victims, with a few exceptions, were inexperienced and separated from their buddies, and generally kept their weight belt on to the bitter end. Several of those who wore a buoyancy vest failed to inflate it, in one instance because of a tear present before the dive commenced. In this series the water depths were shallow and several deaths occurred after "completing" the dive, at the surface. Of a particular interest is the unusual number of victims who had a history of asthma, though this was not necessarily a factor in their demise. Significant cardiac disease was present in one victim but it is not known whether any routine medical fitness assessment would have discovered its presence. In the case of the unfortunate youth with the Wolf Parkinson White syndrome there is much to be said for the advice he received that he should choose to live rather than to follow a cautious fearful existence. Whether scuba diving as such was a critical factor or merely the trigger of the fatal episode is debatable and opinions will reflect each person's philosophy on life.

Of the six breath-hold divers, five were alone at the critical time and none wore a buoyancy vest. Significant factors included fatigue, hyperventilation, epilepsy, a cardiac condition, an asthmatic history, and gross inexperience. Three incidents occurred at the surface and one after leaving the water. It is clear that it is unsafe to allow some asthmatics to undertake prolonged and tiring in-water activities, that even snorkelling may hold dangers. It was only chance which prevented this death occurring during or after his scuba activities.

The scuba divers were alone at the critical time in twelve out of the fourteen cases, either because of separation or because they were diving solo. In the two remaining cases there was nothing more that the respective buddies could have done as water power was irresistible in one case and the cardiac problem probably unsurvivable except in a hospital setting in the other. Once an incident occurred buddies reacted but by this time there had developed an irreversible situation. It is apparent there are still many who fail to accept the self discipline of buddy-diving procedures, and there are graves to prove it. Readers may care to consider what influence a buddy could have had if nearby at the critical time. If it should be decided that the outcome would probably have been the same, consider what other changes in the dive protocol would have resulted in survival, in a danger-free dive.

That three instances occurred in 1984 where totally scuba-ignorant persons were taken for a sea dive by friends is an indication that diving education had not reached everybody. There can never be a method of completely preventing such tragedies, but a well publicised action for damages by a widow or other relative might work wonders.

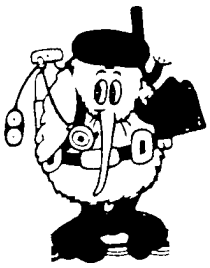
The cheering fact in this report is the absence of deaths among those who were trained, experienced and following the generally accepted guidelines for safe diving. Consider this well.

ACKNOWLEDGMENTS

This report would not have been possible with out the generous support given by the New Zealand Department of Justice and the New Zealand Underwater Association. There has also been valued information provided by other persons.

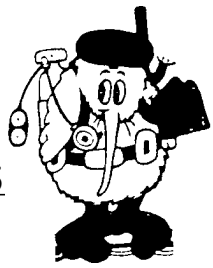
These deaths are among those referred to in the summary "Diving Fatalities in New Zealand 1982 - 1985" on page 22 of the SPUMS Journal 1985: 15(4).

Dr D Walker's address is 1423 Pittwater Road, NARRABEEN NSW 2101, Australia.



DIVING KIWIS

PAPERS AND ABSTRACTS FROM THE NEW ZEALAND CHAPTER OF SPUMS MEETING 7 TO 9 NOVEMBER 1985 KAIKOURA ISLAND, GREAT BARRIER ISLAND



ROYAL NEW ZEALAND NAVY CHAMBER TREATMENTS 1984 and 1985

Peter Robinson

SUMMARY

In 1984 six cases of decompression sickness (DCS) and six cases of pulmonary barotrauma were treated in the recompression chamber at HMNZS PHILOMEL and at the RNZN hospital. Five of the cases of decompression sickness and three of the cases of pulmonary barotrauma had done dives shallower than 21 m (70 feet). One person developing DCS and three with pulmonary barotrauma had dived deeper than 21 m. There were 11 males and one female in this group of damaged divers. Their ages ranged from 16 to 37.

1985 was a busier year for the chamber and the hospital. By 7 November 1985, the day the conference started, 17 males and one female had been treated in the chamber and four other people with mediastinal emphysema had been seen but not recompressed. The diagnoses of those treated were DCS in eight, arterial gas embolism in 9 and one had a presumed coronary occlusion at 33 m (110 feet). Of those who developed DCS only one had not been deeper than 21m. He had only been to 18 m (60 feet). Five of the arterial gas embolism cases had been deeper than 21 m and four shallower. The ages ranged from 15 to 49.

Enquiries about possible diving accidents reach the hospital at least once a week. The chamber is used to treat civilians about once a fortnight and approximately once a month there is a diving death, unrelated to the previous contacts, which never reaches the chamber. This is a depressing commentary on the attitude to safety of New Zealand divers.

Surgeon Commander P Robinson's address is the Naval Hospital, Devonport, AUCKLAND.

POSTSCRIPT

In the month 7 November 1985 to 6 December 1985 five more people required recompression, four males and one female. Two developed DCS, one had dived deeper than 21 m, and three had pulmonary barotrauma. Of these two had dived to less than 21 m and one deeper. Their ages ranged from 24 to 49. In the same period there were two diving deaths in the Auckland area.

HAEMOSTASIS, DECOMPRESSION SICKNESS AND MIGRAINE

JF Cleland

INTRODUCTION

There is no doubt that decompression sickness (DCS) is largely due to widespread direct and in direct tissue disruption as a consequence of bubble formation and vascular block. There is however, a wide and complex range of patho-physiological events which follow the formation of intravascular bubbles and how much these events influence the severity and course of the disease, is not known. However, it is generally accepted that not all of these subsequent events are protective, and indeed some, particularly activation of the haemostatic process and aggregation of platelets at the blood bubble interface, are almost certain to contribute to the problem.

The evidence for involvement of the haemostatic mechanism in DCS is convincing and in fact one could argue that it would be unphysiological for the haemostasis mechanism, platelets in particular, not to respond to the formation of intravascular bubbles with in addition, varying, direct endothelial and other tissue injury. Actually, it would appear that intravascular bubble formation and endothelial damage are not necessary for platelets to aggregate as ex vivo experiments show that platelets will aggregate with decompression stress alone. In addition to these mechanical events, there are other factors important in the pathogenesis of DCS which could operate through activation of the haemostatic mechanism. These include strenuous exercise to the level of exhaustion, fear and hypoxia. The real question is how important is this triggering of the haemostatic mechanism? Does intravascular coagulation provoked by bubbles lead in itself to further significant ongoing tissue injury? If so, there would be three main implications:-

(i) Anti-haemostatic drugs might be helpful in the management of DCS.

(ii) Such drugs may be helpful in the prevention of DCS.

(iii) People with an already hypersensitive or activated haemostatic mechanism could be recognised who could be assumed to have a high risk of DCS with any given decompression stress.

We have now had a decade of diving experience since it was recognised haemostasis might play a part in DCS and in spite of very significant advances in our understanding of the physiology of haemostasis, particularly the role of prostaglandins in platelet function over this period, the literature contains a surprisingly small amount of published related biomedical research. When I reviewed this subject in 1976, I predicted (on the basis of my haematological bias of course), haematological management would be almost as important as recompression. Antiplatelet drugs would provide cheap, at least partial, protection, and we would be able to recognise with blood tests high risk divers with various clinical conditions and advise them accordingly. In contrast it seems any advances in this area have not been translated into practical benefit for us, either as doctors or divers.

I will review these three main areas in turn.

THE USE OF ANTI-HAEMOSTATIC DRUGS IN THE TREATMENT OF DCS

As far as I am aware no drug or combination of drugs has been shown to be safe and effective for the treatment of DCS by controlled double blind studies in humans. It also seems unlikely that such studies will be forthcoming with the fortunately infrequent occurrence of severe DCS usually in remote locations. The usual consequence is delayed treatment which in itself, with established and irreversible ischaemic tissue injury, gives any anti-haemostatic drug little chance of providing benefits. In addition, there are obvious potential hazards in the use of such drugs. Particularly bleeding which must be weighed against their potential benefit.

Dextran

Apart from its obvious potential benefit as a volume expander both dextran-40 and dextran-70 would be expected to have an additional benefit as the result of their antiplatelet action. In spite of this, there seems to be no evidence that dextran therapy has any advantage over simple fluid replacement.

Heparin

Heparin cannot yet be considered to be of predictable consistent value in the treatment of DCS. A few individual case reports suggest a significant benefit and there are a number of animal experiments in which heparin has reduced mortality from decompression sickness. However, other studies, in experimental animals, particularly dogs, have failed to confirm the benefits and concern has been expressed at its use as it is suggested that haemorrhage may play an important role in the pathogenesis of inner ear decompression sickness. In causing haemorrhage into ischaemic areas, particularly the spinal cord, it could aggravate neurological deficits. As I understand it, heparin is only recommended in severe cases of pulmonary DCS unresponsive to recompression therapy.

Antiplatelet Drugs

The role of antiplatelet drugs in the treatment of DCS remains uncertain. There appear to be very few studies in which antiplatelet agents have been used to treat decompression sickness. Prostacyclin, nature's most potent, though short-acting, inhibitor of platelet aggregation, has now been available for approved experimental use in humans for some years. In addition to its potent anti-aggregatory effect, it is also a potent vasodilator, and might be expected to be of benefit in DCS, particularly if used early. I have found only one reference to its use when it was used in association with indomethacin and heparin in dogs with experimental central nervous system ischaemia after air embolism. Neuronal recovery was promoted. Prostaglandin E1 has been used for therapy of DCS in dogs without beneficial effect.

THE ANTI-HAEMOSTATIC DRUGS IN THE PREVENTION OF DCS

Heparin

Studies on the prophylactic use of heparin in experimental animals have been contradictory, some

showing significant protection others showing no benefit. Owing to differences in administration, subcutaneous or intravenous and the timing of the treatment, this drug is not really a practical prophylactic in the human.

Oral Anticoagulants

There is little experimental support for use of prophylactic oral anticoagulants, which have not been found to have a consistent protective effect in experimental animals.

Antiplatelet Drugs

Because of the ease of administration, prolonged affect, safety and low cost, antiplatelet drugs are an attractive proposition for the prevention of DCS. Drugs which have been investigated include aspirin, dipyridamole (Persantin), aspirin in combination with dipyridamole, and aspirin has also been used in combination with levodopa.

Aspirin

Some studies in man have been unencouraging, in showing little protective benefit as measured by platelet survival times and post dive platelet levels. Aspirin given shortly before a dive has also been shown to have no affect on occurrence of DCS. Prophylactic aspirin has also been shown to have no effect in protecting rats from DCS. However, pre-treatment with aspirin for thirty days has been shown to significantly decrease clinical signs of serious forms of DCS in rats, and when used in combination with levodopa this benefit was even more striking. This combination of drugs reduced the mortality of rats from 31% to 5.6%. It may be that aspirin could have a significant benefit if used in an appropriate low dose for a longer period before diving. Although the risks of aspirin ingestion are very small, there remains some theoretical concerns about its widespread use. The prevalence of minor haemostatic disorders, such as Von Wille brands disease, in the community is known to be much higher than previously recognised and it is known in such patients who have a minor or negligible haemostatic defect aspirin can significantly aggravate the problem. Such patients could be at excess risk in diving, particularly in bleeding from mucosal surfaces, eg. nasal passages with mask squeeze etc.

Dipyridamole (Persantin)

Used alone, little practical benefit has been demonstrated even on platelet kinetics let alone clinical signs of DCS.

Aspirin and Dipyridamole

Used in combination, these drugs have been shown to have a synergistic effect on platelet function and this had led to their use in combination in a number of clinical settings, eg. prosthetic heart valves. This combination has been shown to eliminate the immediate post dive fall in platelet survival time but whether this necessarily implies a benefit is uncertain. Other drugs, dipyridamole derivatives, have been used prophylactically in human divers and shown to prevent a post decompression fall in circulating platelet count. Prophylactic use of these drugs has also been shown to significantly decrease the incidence and severity of bends in rats compared with unprotected controls. Once again the significance of these observations for the prophylaxis of clinical DCS in the human remains uncertain.

RECOGNITION OF HIGH RISK GROUPS FOR DCS

Many clinical groups are known to have excess risk of decompression sickness for given decompression stress. These include women, who have more than three times the risk of males, obese people, and unfit and older divers. Whether this excess risk in any way relates to haemostatic variables is not known.

There are an increasing number of disorders being recognised in which there are specific inherited or acquired abnormalities in the haemostatic mechanism either involving platelet function, coagulation factors, coagulation inhibitors or components of the fibrinolytic system. Such disorders, “thrombophilias”, are characterised clinically by a tendency to thrombosis, usually venous, less commonly venous and arterial, and these disorders include antithrombin III deficiency, protein-S deficiency, protein-C deficiency, heparin co-factor II deficiency, and the presence of the lupus anti-coagulant. Collectively however, these disorders are still extremely uncommon and the diving medical could well identify such patients from the past or family history of thrombotic problems, recurrent abortions, etc. On theoretical grounds such patients would be at excess risk of DCS given any decompression stress, but to identify them routine detailed tests of haemostasis could not be justified.

ORAL CONTRACEPTIVES

Thrombotic complications of oral contraceptives have lessened considerably with the use of preparations containing smaller doses of oestrogen or progestogen only preparations. A few studies of female divers taking oral contraceptives have been reported, and no excess risk of clinical DCS has been shown. On theoretical grounds at least however, I believe would be most unwise for a woman on oestrogen-containing oral contraceptives to engage in “aggressive” diving.

MIGRAINE

It has been generally accepted that migraine can be aggravated by diving. It is also generally accepted that migraine is a relative or absolute contra-indication to diving for the reason that its associated neurological symptoms and signs overlap with those of DCS creating confusion in diagnosis.

Documentation of the association of migraine headache with diving is hard to find. In 1944, one report describes 155 medical and university students who were exposed to low pressure, 30,000 to 38,000 feet in hypobaric chambers. Sixteen of these students reported scotomata and headache, and out of these 16, 11 had a history of previous migraine. It was concluded that people with migraine are more likely to get headaches when they undergo barometric change. A subsequent study in 1965, also involved medical personnel, three doctors and one nurse who were subjected to compression to 66-135 feet below sea level. All four experienced scotomata and head ache and developed abnormal EEGs, and of these four, three had a history of previous migraine.

By what mechanism could migraine be precipitated by diving? The evidence is that the association almost certainly has its base in platelet behaviour.

Following the early observation that platelet micro-aggregates are present in the circulation of patients with migraine headache, evidence for abnormal platelet function in patients with migraine emerged during the early 1970's. Initially there was uncertainty as to whether these micro-aggregates were the result of migraine or a major or contributing cause. It has now been shown that such platelet micro-aggregates are also present in the prodromal periods of migraine. And between migraine attacks platelets have been shown to be abnormally sensitive to platelet aggregants such as 5HT and ADP and in addition, platelet enzyme defects have also been reported in migraine sufferers. When platelets are activated and they aggregate in vivo, the contents of the beta granules are released, the so called "release reaction", and this includes the release of beta thrombo-globulin and platelet factor IV. Patients with migraine, both in between and during attacks, have been shown to have increased beta thrombo-globulin levels with the highest levels during attacks. Not all patients, in fact 25% of patients only in one study, show increased beta thrombo-gobulin levels and it seems that patients with classic migraine are more likely to show evidence of abnormal platelet turnover (90%) between migraine attacks than patients with common migraine (33%). These observations of abnormal platelet sensitivity causing or at least predisposing migraine, soundly base the use of aspirin or other antiplatelet approaches to migraine prophylaxis. The first of these studies was reported in The Lancet by O'Neill and Mann in 1978. This showed that prophylactic use of aspirin had a beneficial effect on many but not all migraine sufferers, reducing both the number and the severity of attacks. A subsequent study by D'Andrea and associates reported in Stroke in 1984 adds further support to the likely benefits of aspirin given prophylactically. They showed that administration of aspirin to patients with common and classic migraine who were shown to have in creased levels of beta thrombo-globulin and platelet factor IV both between and during attacks, resulted in significant reduction in levels of both. Similar observations have been made by other investigators.

In conclusion then, it would seem that there is reasonable evidence to base the suggestion that the association of migraine with diving is due to platelet over-reaction to decompression stress with or without bubble formation. It would also seem reasonable to assume that migraine sufferers may be at increased risk of DCS because of their haemostatic "over reactivity".

Although aspirin and similar antiplatelet drugs have not been shown to have a convincing benefit in protecting against DCS in humans, the use of aspirin in such an identified subgroup as migraine sufferers might be expected to show more benefit. Of course, such studies are not likely to be done.

PERSONAL VIEW

I was introduced to diving at Miner's Head, Great Barrier Island, New Zealand in 1972. My early diving experiences were unforgettable but made more so by the fact that almost without exception and even with very conservative, 30 feet scallop dives, I would suffer migraine. Other than with diving, I would normally have suffered no more than two migraine headaches a year. In 1976 I began taking aspirin in the form of Palaprin Forte, beginning 1-3 days before expected diving excursions. This has been completely effective in preventing post dive migraine headache. On occasions I have not taken aspirin and more often than not, I have developed a severe headache. However, in recent years I have become a less adventurous diver, preferring the lesser risks and discomfort of shorter and shallower dives.

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Dr JF Cleland's address is the Pathology Department, Green Lane Hospital, Auckland, New Zealand.

LETTERS TO THE EDITOR

Duke University Medical Center
Durham, North Carolina 27710

29 April 1986

Dear Sir

I have been recently appointed Chief Editor of the new Journal of Hyperbaric Medicine, published by the Undersea Medical Society, starting this year, 1986. This journal will enable medical practitioners, researchers, and other professionals in the field of hyperbaric medicine to keep abreast of current scientific research in this specific area. I am soliciting now for original contributions focusing on clinical application of hyperbaric oxygen (HBO), oxygen effects on body metabolism, treatment protocols, and protectants against oxygen toxicity.

In addition to original research and clinical communications, the journal will carry reviews, technical and preliminary notes, abstract of the literature, letters to the editors and book reviews.

I would be grateful if you would print this letter so that members of SPUMS who might be interested in submitting a contribution know where to send it.

Manuscripts should be submitted to:

Elaine C Frost
Managing Editor
Undersea Medical Society, Inc.
9650 Rockville Pike,
Bethesda, Maryland 20814
USA

Yours sincerely

Enrico M Camporesi, MD
Professor of Anesthesiology
Assistant Professor of Physiology
Director, Clinical Services
The Hyperbaric Medical Center

PROBLEMS WITH MEDICAL CERTIFICATES

Diver Instruction Services
12 Waratah Avenue
The Basin VIC 3154

7 April 1986

Dear Sir

I enclose copies of recent medical certificates supplied by students of our dive school. The names of all concerned have been omitted and the students have granted permission for publication.

I am concerned that many students are being passed, or should I say not being failed on a diving medical if their fitness is questionable. It appears to me that the decision about fitness to dive is therefore being passed to the instructor, and the student.

The Certificate for student A reads: "... has been examined by me for fitness for training in SCUBA diving .... is physically small and of light build. He is healthy and normal for his age and weight but he could expect to have problems in any but the most protected environment, or if he used equipment inappropriate to his size and strength. However, in a sheltered area with 'hand-holding' supervision, it could be possible to train him in underwater activities when he is completely well."

In this case, if the student is taught in a totally "protected environment", in "calm conditions", and a "hand-held situation", what is he going to learn? What happens once he completes training and is turned loose into a normal diving situation? When the student is "completely well" relates to his asthmatic condition!!!

The certificate for student B reads, "... has been examined by me for fitness for SCUBA diving. ... has a medical problem which could at times make it unwise for her to undertake underwater activities, but at other times when she is completely well, she could with due care and in appropriate circumstances participate in SCUBA activities."

We again have the situation where the student can dive if "completely well", ie. no recent asthma activity. On querying the doctor concerned, the "due care" relates to a "one on one, hand-held teaching situation", and "appropriate circumstances" relate to "no current, calm conditions and shallow water". The doctor also stated that all students should only ever be taught on a one to one ratio and that his duty to the student is not to pass or fail, but merely to advise.

If the job of the instructor is to educate and train the student, and eventually "wean" them into becoming an independent, competent and confident diver, how can this be achieved given questionable medical status, and only ever training in perfect conditions? With training in such a tightly controlled situation the student cannot learn independence.

Given the average conditions in Melbourne, where unfortunately you can never get "perfect" conditions all the time, surely we should train the student to cope with the average conditions. If the student must be "hand-held" throughout training, I strongly query whether they should be trained.

The "medical problem" of student B was not stated and would not be discussed by the doctor on "ethical grounds". What of the ethics of letting the instructor take a potential time bomb into the water without the full knowledge of the disability and what the implications of it may be? To overcome the legalities of divulging this private information to the unsuspecting instructor, may I suggest an information release clause on the medical sheet allowing the doctor to discuss any medical history he may feel relevant with the instructor concerned.

Whilst it may appear that I feel that all students should be failed medically if not an Olympic champion, what I am actually asking for is more specific information from doctors to be given to the instructors in questionable cases. I believe that the decision has got to be the doctor's, not the student's, on whether they do or do not dive.

NB: The parents of student A were quite concerned that the medical certificate was very vague. It actually made them concerned that, if their son could only be taught given the strict conditions listed, "what is the use of training him".

Yours faithfully,

David Wailes

DIVER INSTRUCTION SERVICES
FAUI NO. 390

THE SAFETY SAUSAGE

62 Galway Street
Invercargill
New Zealand

11 April 1986

Dear Sir

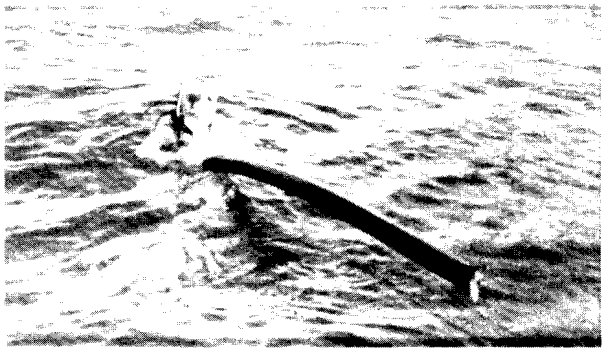
In his paper on Diving Accidents (SPUMS J. 1986; 16(1): 27-30) Dr John Knight recommended as a final article of safety equipment an expensive flare. I agree that this is the most visible safety aid but as an average sports diver I would baulk at the cost.

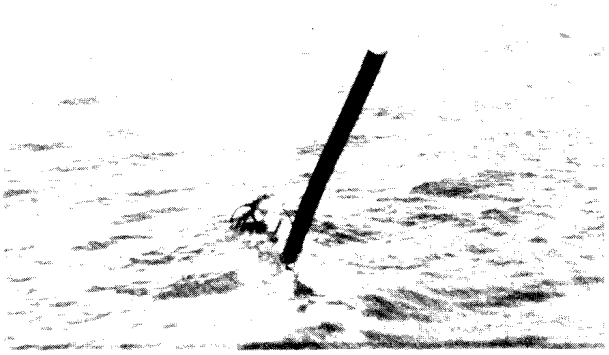
The Safety Sausage or DIT (Diver's inflation tube) is an example of Kiwi ingenuity designed in an effort to overcome the problem of the cost of flares. It was presented at the New Zealand Underwater Association AGM in 1985. It is a red plastic tube 3.2 m by 0.165 m uninflated, and is easily carried in a buoyancy compensator pocket. When a diver surfaces the DIT can be held over the regulator, the purge valve depressed, and immediately he has a long easily visible marker enabling a watching boat to spot him quickly.

This was developed during SAR exercises by the Otago Underwater Club in Dunedin. It can float on the surface for a plane to spot, or by holding with a straight arm down under the water it will act like a flagpole.

I hope SPUMS will help promote it.

Yours faithfully
Judy Johnston





The safety sausage or diver's inflation tube (DIT) fully inflated.

SPUMS NOTICES

OBJECTS OF THE SOCIETY

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

MEMBERSHIP

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

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

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

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

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

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

The requirements for the Diploma of Diving and Hyperbaric Medicine are

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

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

Applications should be directed to the Secretary of SPUMS:

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

SPUMS JOURNAL

Instructions to Authors

Contributions should be typed in double spacing, with wide margins, on one side of the paper. Figures, graphs and photographs should be on separate sheets of paper, clearly marked with the appropriate figure numbers and captions. Figures and graphs should be in a form suitable for direct photographic reproduction. Photographs should be glossy black and white prints at least 150 mm by 200 mm. The author's name and address should accompany any contribution even if it is not for publication.

The preferred format for contributions is the Vancouver style (Br Med J 1982; 284: 1766-1770 [12th June]). In this Uniform Requirements for Manuscripts Submitted to Biomedical Journals references appear in the text as superscript numbers.[1-2] The references are numbered in order of quoting. The format of references at the end of the paper is that used by The Lancet, The British Medical Journal and The Medical Journal of Australia. Page numbers should be inclusive. Examples of the format for journals and books are given below.

1 Anderson T, RAN medical officers' training SPUMS J 1985; 15(2): 19-22.

2 Lippmann J, Bugg S. The diving emergency handbook. Melbourne: JL Publications, 1985.

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

Measurements should be in SI units. Non-SI measurements can follow in brackets if desired.

Reprinting of Articles

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

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For further information write to
SPUMS
80 Wellington Parade
East Melbourne VIC 3002
Australia.

ELECTION OF OFFICE BEARERS

Nominations are called for the positions of President, Secretary, Treasurer, Editor and three committee members.

Each nomination is to be proposed and seconded and countersigned by the nominee.

Nominations are to be in the hands of the Secretary of SPUMS (Dr David Davies, Suite 6, Killowen House, St Anne's Hospital, Ellesmere Road, Mount Lawley WA 6050) by 16 August 1986.

CONSTITUTIONAL CHANGE

The Executive Committee consider that the Chairman of the New Zealand Chapter of SPUMS should be an ex-officio member of the Executive Committee.

If this proposal is accepted the Executive Committee will consist of the seven directly elected office holders, the Past President, and the Chairman of the New Zealand Chapter of SPUMS.

Any member objecting to this proposal should notify the Secretary of SPUMS (Dr David Davies, Suite 6, Killowen House, St Anne's Hospital, Ellesmere Road, Mount Lawley WA 6050) by 16 August 1986.

If there are no objections the proposal will be deemed to have been carried and the constitution altered.

If there is an objection a postal ballot will be held.

NEWS FROM NEW ZEALAND

Dr Warren Paykel, the returning officer for the elections for the New Zealand Chapter, has had only one nomination for the position of Chairman, Dr Allan Sutherland, and only one for the position of Secretary, Dr Peter Chapman-Smith. These gentle men are declared elected to their respective positions.

Dr Sutherland's address is
"Outspan"
Bush Road
Albany RD1
Auckland

Dr Peter Chapman-Smith's address is
67 Maunu Road
Whangarei

Besides being Secretary of the New Zealand Chapter Dr Chapman-Smith is Treasurer of the Chapter. All New Zealand members and associates are requested to forward their annual subscriptions, with the subscription reminder, to Dr Chapman-Smith at the above address. He will remit the New Zealand subscriptions, in Australian dollars, to the Treasurer of SPUMS, Dr Grahame Barry. This will save New Zealand members and associates the inconvenience of obtaining a cheque in Australian dollars to cover their subscription to SPUMS.

The Second Conference of the New Zealand Chapter of SPUMS will be held at Whangamata from 13 to 16 November 1986. The conference organiser is Dr Mark Fraundorfer, whose address is PO Box 56, TAURANGA.

Dr Harold Coop has designed a logo for the New Zealand Chapter of SPUMS. It is reproduced below.

DOCTORS WITH TRAINING IN UNDERWATER MEDICINE

We publish below a list of current members of SPUMS resident in Australia, who have completed at least the Royal Australian Navy School of Underwater Medicine introductory course, or who have notified the Secretary, as requested in the SPUMS Journal (1985, 15(2): 3.) that they have had equivalent training. The list has been compiled with the co-operation of the School of Underwater Medicine (SUM) and includes all members that can be identified from the SUM records, as well as those who wrote in. As a result it may include doctors who no longer do diving medicals. The addresses given are those to whom the Journal is sent and so may not be their professional rooms. Those who have had equivalent training and whose names are not in this list are asked to write to the Secretary of SPUMS giving details of their training.

New South Wales

Dr JM Anderson
91 Donnison Street
GOSFORD NSW 2250

Dr T Anderson
School of Underwater Medicine
HMAS Penguin
BALMORAL NAVAL PO NSW 2091

Dr C Edmonds
25 Battle Boulevard
SEAFORTH NSW 2092

Dr C Finlay-Jones
165 Morgan Street
MEREWETHER NSW 2291

Dr R Gray
21 Coombar Close
COFFS HARBOUR NSW 2420

Dr R Green
47 Shorter Avenue
BEVERLEY HILLS NSW 2209

Dr JT Horgan
232 Mona Vale Road
ST IVES NSW 2075

Dr P Nolisch
33 Mann Street
NAMBUCCA HEADS NSW 2248

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

Dr C Lowrey
233 Raglan Street
MOSMAN NSW 2088

Lcdr CJ McDonald
6/31-39 Elamang Avenue
KIRRIBILLI NSW 2061

Dr W Pettigrew
C/- Lidcombe Hospital
LIDCOMBE NSW 2141

DR CP Pidcock
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NELSON BAY NSW 2315

Dr F Summers
56 Hickson Street
MEREWETHER NSW 2291

Dr I Unsworth
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Dr NJ Wishaw
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65 Ports Street
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MACKAY QLD 4740

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Dr P Sullivan
33 Rutledge Street
COOLANGATTA QLD 4225

Dr RL Thomas
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Dr M Unwin
8 Fulham Road
PIMLICO QLD 4810

Dr RM Walker
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SOUTHPORT QLD 4215

Dr J Williamson
137 Wills Street
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Royal Hobart Hospital
HOBART TAS 7000

Dr P McCartney
PO Box 1317N
HOBART TAS 7001

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8 Ascot Street North
BALLARAT VIC 3350

Dr J Knight
80 Wellington Parade
EAST MELBOURNE VIC 3002

Dr C Lourey
25 Hastings Road
FRANKSTON VIC 3199

Dr JE Mannerheim
22 Frank Street
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Dr I Millar
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1 Chickerell Street
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Dr R Moffitt
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Dr J Silver
57 Electra Street
WILLIAMSTOWN VIC 3016

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Western Australia

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RAP Special Air Services Regiment
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MOUNT LAWLEY WA 6050

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

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

DIVER EMERGENCY SERVICE
008-088-200

From 2 July 1986 there will be a new telephone number for the Diver Emergency Service (DES). The toll-free number is 008-088-200 at all times.

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

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

The new arrangements allow for easier access to advice for diving casualties,

THE NEW NUMBER FOR DES IS

008-088-200

NEW ZEALAND CHAPTER OF SPUMS
FIRST AGM AND SCIENTIFIC MEETING

To be held on the 13, 14 and 15 November 1986 in Whangamata, New Zealand.

All SPUMS Members, both New Zealand and Australian, are welcome.

The Conference will commence on Thursday 13 November, with initial Registration, followed by the first AGM of the New Zealand Chapter SPUMS. Scientific Papers will be given in the afternoon.

Conference Delegates will have the option of booking into the Cedarwood Motor Inn which has 26 fully self-contained units and all holiday resort facilities. On the Thursday evening following cocktails a barbeque and tennis tournament will be conducted on floodlit tennis courts, weather permitting.

On Friday 14th, weather permitting, a chartered boat will take divers out to Mayor Island for one of two possible dives at that location. It will be possible for people to go ashore on Mayor Island and spend the afternoon walking the island's various tracks. This locality is a mecca for big game fishing in the North Island. A late afternoon return to Whangamata will be followed by a Seafood Smorgasbord (with luck) dinner with a short scientific meeting to follow.

On Saturday morning the chartered launch departs for further dives in local dive spots, with lunch on board. Alternative activities will be made available for non-diving delegates or spouses who choose not to dive.

A free session will be made available on Saturday afternoon to be followed by further papers between 6 pm and 7.30 pm. Dinner and a Cabaret evening conclude the formal aspect of the Conference on the Saturday night leaving a further half day on the Sunday for those who want to take the opportunity for a further dive.

For further information on the programme or registration please write (airmail) to Dr MR Fraundorfer, PO Box 56, Tauranga, New Zealand.

PROJECT STICKYBEAK

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

Dr D Walker
PO Box 120
NARRABEEN NSW 2101

ARTICLES OF INTEREST FROM OTHER JOURNALS

WHY AMERICAN DIVERS DIE

INTRODUCTION

The National Underwater Accident Center (NUADC) at the University of Rhode Island annually publishes a report analyzing and describing diver fatalities from a previous year. Undercurrent has subsequently published a synopsis of this report so that divers may better understand what causes accidents and fatalities and then apply the sad lessons learned from these deaths to their own safe diving practices.

The most current NUADC report covers the 1982 calendar year; we take the responsibility for any inadvertent errors due to our editing of the material. We also would like to thank John McAniff, Director of NUADC, for his diligent pursuit of data, his careful analysis, and his co-operation in getting this material into the public domain where these cases contribute to safe diving and, in fact, save lives.

THE REPORT

The NUADC is happy to report that during 1982 we recorded the lowest number of fatalities for any year since the beginning of this study in 1920. 102 casualties were recorded, including 74 non-occupational fatalities, 10 skin (breathhold) diving, and 18 occupational fatalities. In contrast we recorded 187 fatalities during 1974.

We estimate that as of December 1982, approximately 5.25 million persons have been trained by the major agencies. By making allowances for dropouts and cross-certifications we estimate the US active diver (one who dives at least three times per year) population at approximately 2.45 million to 2.6 million, suggesting a fatality rate during 1982 for non-occupational US underwater divers was between 2.84 and 3.02 fatalities per 100,000 active divers.

Florida had 15 fewer fatalities in 1982 than in 1981. 17 divers lost their lives in Florida caves in 1981; in 1982, only three cave deaths were recorded. California, with only 14 underwater diving fatalities for 1982, has recorded its safest year since the inception of this research. There were 20 deaths in 1981 and 36 in 1973. The state of Washington recorded 17 deaths in 1979 as their peak, while only recording 9 fatalities in 1932.

Environmental Aspects of Non-occupational Fatalities

During 1982, 63 fatalities occurred in oceans, bays or seas; lakes, ponds, and sloughs accounted for 5 deaths; one death occurred in a man-made rockpit; two others lost their lives while diving in rivers; one death occurred in the Great Lakes, and three in caves.

With one exception, all of the 1982 deaths occurred in waters shallower than 120 feet. The exception was a diver off the New Jersey coast on a well known wreck at a depth of 170 feet.

As in previous reports, the NUADC will again stress that some insurance companies unfairly choose to rate scuba divers if they routinely exceed a depth of 50 feet. Some companies have gone so far as to double or triple the premium if this depth is exceeded, while other companies have simply chosen not to insure scuba divers. Our studies have shown that there is no basis for this 50 foot depth limitation. The NUADC strongly believes that such rating should only be used if the diver routinely exceeds the recognized "safe diving limits," ie. 100 feet or 130 feet if we use the US Navy limits. At approximately three deaths per 100,000 scuba diving is now certainly way below many other sports activities. This fatality rate certainly supports the abolishment of the insurance companies' rating systems.

During 1982, eight cases were cited as involving heavy or dangerous surf and three cases involved strong currents. Three of the eight cases in which heavy or dangerous surf were cited, involved victims who were at the time under instruction. Two of the eight heavy surf cases involved the victim being thrown onto rocks and possibly losing consciousness. One of the current cases involved a diver on his first dive in the ocean who had "been taught by a friend three days ago." This victim was swept away by a very strong outgoing tide.

During 1982 there were 34 fatalities while diving from shore, 10 fatalities while diving from a charter dive boat, 12 while using a private vessel, and California and Florida each recorded three charter boat fatalities; the US Virgin Islands, the states of Washington and New Jersey, and the Bahamas recorded one each.

Age Experience and Training

Eleven of the 74 non-occupational underwater diving fatalities were females; seven were 26 to 30 years old. Thirty-six of the 63 male victims were between 21 and 35, and just over 10% of the male victims fell into the age group of 46 to 50.

The experience of non-occupational underwater diving fatality victims is shown in Table 1. The three first listed levels of experience probably indicate a minimal number of dives per diver, while the 4th, 5th and 6th levels probably indicate dozens to hundreds of dives per victim.

Fatalities During Training

Eleven fatalities occurred during training in 1982. One of the Washington State training cases was a 49 year old man who had apparently suffered a myocardial infarct a few days prior to this dive, which was to be his final certification dive. In the second, a 30 year old female was caught in a sudden, violent squall and died of a massive air embolism. This was also the final certification dive.

Both California cases occurred on the rocky shores of Monterey County. A 28 year old female panicked when she was swept into a cove by very heavy surf. The following day a 41 year old died a few miles south of the first incident, suffering a myocardial infarct while in ten feet of water.

The Florida case claimed a 54 year old male who suffered a massive cerebral air embolism when he made an ascent from 30 feet with his vest inflated.

TABLE 1
Experience of Non-occupational Underwater Diving Fatality Victims, Yearly, 1970-1982

Percentage of cases in each category

Experience	1970	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982
First dive ever with scuba	11	16	8	4	11	8	10	10	9	6	11	4	12
First dive in open water	6	7	4	5	6	6	5	1	1	10	4	3	5
Early open-water	31	24	21	34	37	25	30	26	34	21	37	23	20
Some experience	33	19	37	16	24	20	34	40	45	39	33	32	23
Considerable experience	13	23	14	21	16	28	16	19	7	18	12	26	17
Very experienced	6	11	16	10	6	13	5	4	4	6	3	12	23

A 61 year old male suffered a cardiovascular event while in a one-day training course in the Virgin Islands.

The Rhode Island fatality during training involved a 30 year old male engaged in what was reportedly an advanced scuba diving class. He had been ill early in the day but felt well enough to dive. When approximately 200 feet off shore, with full scuba gear but breathing on a snorkel, he apparently got into difficulty and with the assistance of a buddy was hauled up on rocks. When the instructor reached him he was unconscious. He died about two hours later at a local hospital. The autopsy found nothing other than asphyxiation due to drowning.

An 18 year old girl lost her life in a Texas lake while diving with her instructor and two others. Her buddy said that she had gone deeper than 40 feet and that he began to follow her bubbles down when all of a sudden they stopped. The depth was 110 feet and the water murky, with extensive entanglement possibilities with old trees, brush and snagged fish lines. The victim's body was found eight days after the accident.

One mile off Freeport, Grand Bahama Isle, a 43 year old female in formal diver training apparently suffered an embolism. Upon autopsy, it was determined that she suffered a ruptured ovarian cyst during the dive, which may have caused great pain and the resulting embolism and drowning.

During 1982 two fatalities occurred while the victim was receiving instruction from a person not qualified or certified to teach diving. The first case occurred when the victim and the victim's brother and his friend were to introduce the victim to scuba equipment for the first time. On the first two dives, the victim experienced difficulty with his ears and also had a problem with his buoyancy compensator. The two brothers apparently made a dive to a depth of 70 feet and returned to approximately 30 feet where the victim was left alone to swim to shore. The victim's brother and his friend then met at 40 feet and made another dive to 70 feet. Upon returning, they found the victim in 25 feet of water his face mask off and his regulator out of his mouth. The victim was towed to shore without any effort to drop his weight belt or inflate his buoyancy compensator. The victim had suffered a massive embolism.

In the second incident, a 41 year old male, while diving with the would-be instructor in ten feet of water, became tired and wanted to go to shore. The two started to shore on the surface. The buddy instructor reached the shore first, looked back and could not locate the victim. The autopsy showed that the victim had experienced a myocardial infarct several days before his death.

A review of the nine formal training fatalities reveals some interesting points. Three suffered a cardiovascular event which could have been prevented had a proper physical been done on each of these persons. The same is probably true of the young lady who died as the result of the ovarian cyst. Another victim was reported ill that morning, but was still allowed to dive. The young lady who died in a Texas lake simply went deeper than she should have. In at least two cases, heavy surf or a sudden squall were contributory to the victim's demise.

The NUADC has frequently mentioned the need for medical histories of potential diving students. This is even more important for persons over 35 years of age when potential exists for cardiovascular problems.

It is absolutely essential that students in a formal diving course be given very close supervision. We have recorded many cases in which the student has been allowed to leave the group to return to his base of operations either alone or in the company of an equally untrained buddy. There seems to be a definite need to develop a special category of safety assistant or nurse-maid to assist the instructor in his open-water program. And once again we caution instructors to be very much aware of environmental conditions such as heavy surf, fast currents, and poor underwater visibility. Each contribute to several deaths a year.

CAVE DIVING FATALITIES

The most notable reduction of fatalities in any single kind of diving is found in cave diving, which contributed 25 fatalities in 1974. In 1981 the total was 17 fatalities, but 1982 had only three, all of which occurred in Florida.

One incident took the lives of two young men who had been warned not to go into the caves, and will be discussed later. In the third cave diving fatality, a 35 year old male entered the cave structure with three partners. All four began to realize that they were running out of air and made an escape from the cave through an open pothole, only to find that a victim had been left behind. His body was recovered several

TABLE 2

Probable Starting Causes of Diving Fatalities, 1976-1982

Estimated Cause	Number of Cases 1976	1977	1978	1979	1980	1981	1982
(A) Medical and Injury Causes							
1. Possible exhaustion, embolism or panic	24	25	24	33	28	12	13
2. Diagnosed air embolism	10	16	12	14	10	8	11
3. Cardiovascular event	8	4	4	5	6	3	4
4. Nitrogen narcosis	1	0	0	2	3	2	0
5. Hit by boat, extensive injuries	2	2	2	3	1	0	2
6. Aspiration of vomitus, etc.	1	2	1	2	2	0	0
7. Possible intoxication	1	1	0	0	3	1	0
8. Possible choking, wad of gum	1	0	1	1	0	0	1
9. Decompression sickness	1	0	1	1	0	0	1
10. Cramps at depth/cold	0	1	0	0	0	0	0
11. Ruptured eardrum	0	0	1	0	1	0	0
12. Ruptured stomach blood vessel	0	0	0	1	0	0	0
13. Gunshot	0	0	0	1	0	0	0
14. Epileptic Seizure	0	0	0	0	0	0	0
15. Asphyxia/regurgitated food	0	0	0	0	0	0	1
16. Possible suicide	0	0	0	0	0	0	1
Total Medical Causes	49	51	45	62	54	27	33
(B) Environmental Causes							
1. Lost or out of air in cave	21	7	11	12	10	17	3
2. High waves or surf	3	4	3	7	1	7	4
3. Strong current	7	2	3	0	1	1	7
4. Entangled in kelp or weeds	6	2	2	3	4	5	1
5. Lost under ice	3	1	3	3	2	6	0
6. Suspected shark attack	1	0	0	0	2	1	1
7. Entangled in external lines/ropes, etc.	3	3	3	3	1	3	0
8. Night dive, lost sight of shore lights or lost buddy	1	0	1	1	0	0	0
9. Foot wedged in rocks	0	0	0	0	1	0	0
10. Sucked into dam gate	0	0	0	0	0	2	0
11. Lost in wreck (silt)	0	0	0	0	0	1	0
Total Environmental Causes	45	19	25	29	28	43	16
(C) Equipment-related Causes							
1. Out of air at depth	7	12	17	6	8	3	3
2. Overweight at depth	1	1	1	0	4	2	1
3. Weight belt entangled-tank/vest straps	1	0	1	0	0	0	1
4. Poor maintenance regulator	2	0	0	2	0	3	1
5. Equipment “tied” on victim	1	0	1	0	1	0	1
6. Tank fell off backpack, neck strap strangled victim	1	0	0	0	0	0	0
7. Accident back-mounted buoyancy	1	0	0	0	0	0	0
8. On anchor line, struck on head by boat	0	1	0	0	0	0	0
9. Tangled in buddy’s dropped weight belt	0	2	0	0	0	0	0
10. Lost buddy line, black water	0	1	0	0	0	0	0
11. Burst safety disc, tank flooded	0	1	0	0	0	0	0
12. Air reserve pull rod under tank band	0	1	0	0	0	0	0
13. Carbon monoxide poisoning, bad air	0	0	1	1	1	0	0
14. BC oral inflator broken	0	0	1	0	0	0	0
15. Regulator freeze-up	0	0	0	1	0	0	0
16. Lost weight belt, rapid ascent	0	0	0	0	0	1	0
17. Dry suit inversion	0	0	0	0	0	0	1
18. Entangled in flag line	0	0	0	0	0	0	1
Total Equipment-related Causes	14	19	22	10	14	9	8
Probable Cause Not Defined	39	13	23	29	13	24	17

hours later. None of the three cave diving victims had any training whatsoever for this kind of diving.

Other Deaths

The single wreck diving fatality involved a 38 year old male with 20 years of wreck diving experience. He frequently dived the wrecks alone, which he did in this case, even though there were many others on board the charter boat he had taken. This victim was found lying on his back in 170 feet of water with the regulator out of his mouth.

Multiple scuba diving fatalities have occurred frequently in previous years. In 1981, there were eight double fatalities and one triple fatality. The only double event in 19S2 happened in a Florida cave, after two men had been warned by an instructor not to enter the cave systems. They had no lines, no flashlights, and were found lost in the silt inside the cave

Several or many divers in the water, diving in a group, may add more confusion than safety. In earlier reports, the NUADC has consistently emphasized the need to use the buddy system, a one-on-one relationship between two divers who are in close proximity, and each looking out for both himself and his buddy.

Starting Causes of Fatalities

Three categories of probable starting causes of non-occupational underwater diving fatalities have been designated: Medical and Injury Causes; Environmental Causes; and Equipment-related Causes.

Medical and Injury Caused Deaths

Under medical and injury causes, "possible exhaustion, embolism, or panic" may include cases which have exhibited panicky behaviour, confusion and disorientation. Also included is the condition described as 'sudden drowning syndrome' (SDS). SDS was first noted by the NUADC several years ago. We have had a number of such cases each year. Typically, they involve a diver who has been to a depth of 50 feet or more in considerably cold water. Upon returning to the surface, he is apparently all right, though he may be shivering. He gives his buddy the okay sign, and they start returning either to their boat or to shore. After a few strokes, the buddy turns to find that the victim is laying face down on the surface, not swimming, totally expired. There has been no outcry, no splashing, and no panic.

In order to understand this new syndrome, the NUADC has consulted many physiologists and hyperbaric specialists. We still do not know the cause. One possibility is that slight hypothermia coupled with the well-known mammalian diving reflex (the slowing of the heart rate upon immersion of the face in cold water), results in a cardiac arrhythmia and sudden unconsciousness, followed by drowning.

Possible exhaustion, embolism, or panic contributed to 13 of the fatalities in 1982, while 11 cases were diagnosed as air embolism. The four cardiovascular events are considered both probable starting causes and the final results of the events.

The 1982 cases include one death caused by decompression sickness, a very experienced 38 year old male who died while diving 170 feet deep on a wreck off the New Jersey coast.

The possible suicide is a 26 year old male who had been using marijuana and alcohol extensively. Friends reported that for a number of weeks this man had contemplated suicide. He disappeared on the third dive of the day in 100 feet of water off the California coast. The 1982 cases include at least five in which drug abuse may have been a contributing factor, including either alcohol, marijuana, or prescription drugs.

Environmental Causes

Sixteen cases may have had an environmental or external factor as a starting cause. Lost or out of air in a cave accounted for three of the 1982 events. High waves or heavy surf accounted for four fatalities, while seven were attributed to extremely strong currents.

One case was determined to have been caused by entanglement in kelp. A 21 year old college student from the state of Washington had completed his basic training courses two months prior to the accident. While engaged in academic research, he swam across a heavy kelp bed on the surface, and became exhausted. He panicked and sank to the bottom in ten feet of water. Efforts at cardiopulmonary resuscitation were unsuccessful. One victim in 1982 disappeared near Elliot Key, Florida, in about 70 feet of water. This victim was not recovered, but pieces of his clothing, including swim trunks and other equipment, were recovered and all showed signs of having been attacked by a shark.

Equipment Related Deaths

For equipment related cases, three divers died because they ran out of air. A fourth diver was severely over-weighted for the depth of his dive. Weight belt entanglement in the victim's vest straps was the cause of a single case in 1982, while poor maintenance of a regulator led to the death of still another diver. A 28 year old male was found off the coast of Maine in an upside down position with the legs of his dry suit hyper-inflated, while the fast moving waters of a river in Michigan claimed the life of a 30 year old male when he became entangled in his diver's flag line and fishing gear on the bottom.

A Washington state Fish and Game Department biologist lost his life while diving for the first time with a dry-suit that had been borrowed. His 40 pound weight belt was not dropped during the incident. It was said that he had completed a 50 foot dive, and on the way to the surface had experienced difficulties, and sank back to the bottom. It was later determined that the filler hose for the dry suit had not been attached, or perhaps had become loose during the dive.

The NUADC only received information on 12 buoyancy compensator devices worn in fatal accidents in 1982. Of the 12, three were not used and found to be faulty afterwards.

Free Diving

Five of the ten skindiving (breath hold or free diving) fatalities took place off Florida shores. Two cases were recorded in Hawaii, while single cases were noted in California, Oklahoma and South Carolina. Six occurred

in ocean waters, while three occurred in either lakes or ponds, and one fatality occurred in a swimming pool.

One male victim, age unknown, was struck by a boat while snorkelling during a diving instruction class in an Oklahoma lake. A 32 year old male drowned in ten feet of water in a pond on a golf course while attempting to recover golf balls in South Carolina. A 63 year old male victim was one of the two Hawaiian deaths which occurred while snorkelling alone from a charter boat. This victim was later found to have suffered cardiovascular failure. The second Hawaiian death was a 48 year old man who swam off alone and whose body was never recovered. A 47 year old US Navy officer, who died skin diving in Florida, may have been the victim of shock as the result of extensive jelly fish stings. In Florida, a 17 year old boy who had been snorkelling from an aluminium canoe in a lake was struck by lightning.

During 1982, the NUADC was able to verify autopsies on 85 per cent of the non-occupational diving fatalities. Eleven of the victims died due to barotrauma (ie. embolism). If proper autopsy protocol had been followed in the 44 "drowning" cases, as many as ten more might have been determined.

Four male victims, respectively 41, 49, 51 and 61, died from cardiovascular problems. Three of the four were undergoing instruction at the time of death. In the category "major haemorrhages/trauma," we have included two cases. In the first, a 43 year old male, was diving in a heavily travelled waterway off the Massachusetts coast when he was struck by a 60 foot vessel as he surfaced. The victim had been diving from an inflatable boat which flew a divers' flag, but was more than 150 yards from the site of the accident. A 53 year old female victim, said to have been very experienced was apparently struck by a propeller while diving from a friend's boat off the coast of Florida. Her body was recovered several hours later with a massive laceration of the forearm. She apparently bled to death before help arrived.

One victim was found to have aspirated a large chunk of hamburger. He apparently choked to death.

OCCUPATIONAL FATALITIES

There were eighteen occupational deaths in 1982 including five US Navy divers who died in a single incident off the Philippines. Newspaper accounts intimated a decompression problem while working with a Navy submarine, but no further details are available.

One occupational victim was a 39 year old, well-trained diver who was operating on surface-supplied air in 40° water. His hoses apparently became severely entangled in wreckage and he was unable to free himself. Though he wore a bail-out bottle, he did not attempt to make the surface with this device. The owner of the salvage vessel for whom the victim was diving recognized the entanglement problem and donned a dry-suit in an effort to help. In his haste he broke the zipper. Tape was hurriedly put around the legs of the suit, but the rest of the jacket was left open. He then donned a scuba tank, jumped over the side and was never seen again. After two and one-half hours below, the first victim ballooned to the surface in an upside-down position, still entangled and trapped below the surface. The cause of death was determined to be acute decompression sickness.

One fatality was a 26 year old civilian diver working on a naval ship in Long Beach, California. On the first dive of the day, the victim experienced a regulator failure and had to buddy breathe to reach the surface. Apparently, the same thing happened on the second dive, but without a buddy nearby to assist him. The victim died of an air embolism.

Another fatality involved a 60 year old victim who had completed a scuba diving course a week before his death. This victim, along with his son, was trying to commercially harvest quahogs in Narragansett, Rhode Island. There was no witness to the actual death, but the body was found on the bottom about one hour after he was first missed.

Two separate fatalities occurred diving for gold in California rivers. In one incident, the victim had reportedly taped his tank to his wetsuit with a criss-cross pattern using fibreglass tape. This tape was also used to fasten some equipment to his weight belt. He apparently attempted to drop the belt, but it fell behind him, causing his feet to rise to the surface and leaving him without air, The body of this victim was not recovered until the following day.

DISCUSSION

In looking at the causes of diving deaths over the years, patterns emerge which the conscientious diver need be aware of if he is to dive safely. Nearly every death is preventable, and in nearly all cases the individual diver is in full control over his own fate. In considering one's own safety, keep these notions in mind.

Panic

Panic is no doubt both a cause of death and a precursor to death. The wise diver will recognize the signs and move to terminate the dive or terminate the panic. Panic may set in for no apparent reason or for plenty of good reasons, such as running out of air, getting lost in low visibility, or having a major equipment problem. In most cases, that kind of panic is preventable by making the right diving decisions.

Bad Pre-dive Decisions

Many deaths are caused when one dives in lousy weather, when two divers go out in a boat and don't leave someone aboard, when inexperienced divers go cave diving, when divers struggle through high surf, or when one goes diving after a night of partying. Problems that occur are obviously avoidable.

Bad Pre-dive Equipment Decisions

Many divers buy the wrong gear and many more fail to maintain it. The SOS decompression meter is virtually useless. Many regulators are ineffective below 100 feet at low tank pressure, especially if the diving conditions are difficult or a diver is working hard or panicking. An excess of equipment is no substitute for good diving skills.

Regardless of what equipment one has, it is not worth a nickel if not properly maintained. Divers have panicked and died because a worn fin strap or a worn mask strap has broken. BC hoses not checked before the dive have come unfastened from the flotation devices, rendering the BC useless. Rusty regulators have failed to deliver air and rusty tanks have clogged

TABLE 3

Non-occupational Underwater Diving Fatalities, Results of Autopsies, Yearly 1970-1982

	Number of Autopsies												
Primary Complaint	1970	1971	1972	1973	1974	1975	1976	1977	1978	1979	1980	1981	1982
Asphyxiation or drowning	25	26	22	32	29	29	39	45	49	61	60	45	44
Barotrauma/Embolism, etc.	9	12	9	8	14	12	10	16	12	17	12	13	11
Injury to head (often plus "drowning")	5	2	2	0	2	0	2	0	2	3	2	0	0
Cardiovascular syndrome	5	1	3	6	5	4	8	2	5	4	6	3	4
Aspiration of stomach contents	3	1	2	1	1	1	0	2	1	2	0	0	1
Acute decompression sickness	0	0	1	1	1	0	0	0	0	1	0	0	0
Intestinal disorder	0	0	1	0	0	0	0	0	0	0	0	0	0
Bilateral ear rupture	0	0	0	0	1	0	0	0	1	0	0	0	0
Gas contamination	0	0	0	0	0	1	0	0	0	1	0	0	0
Major haemorrhages/ body trauma	0	0	0	0	0	0	0	0	0	0	0	0	2
Total	47	42	40	48	53	47	59	65	70	89	80	61	62

the first stage. Uncalibrated depth gauges have led divers ten or more feet deeper than their dive plan without their knowing it.

Bad Diving Decisions

Decisions made in the course of a dive can mean life, death, or serious injury. Too many divers fail to know the tables and let someone else determine their bottom time. Divers exceed the dive plan, dropping down an extra twenty feet or perhaps making a dive to free the anchor although they have already reached the maximum table time.

Some divers proceed with a dive when the conditions get rough and the dive should be cut short. Some divers defer to their buddy's judgement rather than let their own sensibilities and skills determine their own course of action. Some wear too much weight, while others, when in a difficult surface situation, fail to drop their weight belt, perhaps because they do not want to buy another or to admit to their peers they were in trouble.

CONCLUSION

The causes of deaths range from the obvious and common to the unlikely and bizarre. Nearly all, if not all, can be prevented. To survive, one cannot respond to peer pressure or unwise buddy pressure. Safe diving is an individual matter.

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

PATHOPHYSIOLOGY OF DROWNING *

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In view of recent interest in the causes of drowning both in adults,[1] and in children, and the special contributions made by Australian medical scientists to an understanding of the mechanisms of drowning,[2-7] it seems appropriate to review the pathophysiology of drowning itself. Management of the potential near-drowned victim depends on such an understanding.[8,9]

The Submerged Victim

Although death comes quickly and in a very unsubtle way, there are several ways in which subjects die while in the water.[10] Brain death is the end result, but the first link in the chain may be cerebral hypoxia, carbon dioxide narcosis, laryngeal spasm, reflex lung changes, or vagal cardiac inhibition. In a pedantic sense, some define true drowning as death caused by the aspiration of fluid into the air passages. Death caused by laryngeal spasm, reflex pulmonary changes, or vagal cardiac effects might be more correctly termed "death from submersion"[9] rather than from drowning. In freshwater drowning, it has been known for 30 years[11,12] that large volumes of water can pass across the alveolar capillary interface.[13]

The volumes of water inhaled by a submerged victim may be relatively small. Depth is no guide: children, intoxicated and epileptic subjects, and those with cervical and head injuries can drown in very shallow water. In some cases, the submerged victim inhales large volumes of water; however, there is no evidence that the increases in blood volume is beyond the capacity of the heart and kidneys to compensate for this potential fluid over load.[9] The heart compensates for this with the basic reflexes of Starling's law, and

* Based on an address to the Water Safety Conference, New South Wales Government Department of Leisure, Sport and Tourism, held at the Opera House, Sydney, 11 October 1984.

the Bainbridge reflex.

All forensic pathologists subscribe to the theory of "dry drowning" in humans. This is a condition of fatal cerebral hypoxia brought about by in adequate ventilation which is not caused by mechanical blockage of the bronchi by the drowning fluid, usually water, but rather by laryngeal spasm, or deleterious lung reflexes,[2-4] or mechanical blockage by mucus and froth. The frequency of dry drowning is variously estimated to be from 10 per cent to 20 per cent.[1,14] There is good experimental evidence to support the phenomenon of "dry drowning".[9] In the experimental drowning of littoral mammals, such as beavers (with diatom monitoring)[10], it has been shown that not all animals which were trapped fully submerged, and "drowned", actually took water into the lungs.**[15]

When dry drowning occurs, it appears that a small amount of water enters the larynx or trachea, and sudden laryngeal spasm (as a vagal reflex) occurs. It is followed by an immediate outpouring of thick mucus; foam and froth develop, and, in some cases, a physical mucus plug may form. It seems likely that, when the spasm relaxes shortly before death, water may be prevented from entering the trachea and lungs by the foam and froth which act as a physical barrier, and by bronchiolar spasm.[4] Whether or not such lung reflexes are facilitated by a raised blood alcohol level is unknown but it has been suggested as one explanation for the greater number of "dry drowning" cases in adults, compared with that reported in series of child drownings.[16]

The Drowning Episode

When a sober victim first submerges, there is an initial period of voluntary apnoea. From personal observations in infants and toddlers in so-called "drown-proofing" classes (well meaning, but misguided in my view), one is struck by the failure of an infant to struggle as the head goes below the surface - usually the child simply holds his breath; makes automatic, but ineffective, paddling type movements; and calmly sinks to the bottom. It is almost certain that the same pattern is followed in the accidental drowning of children.

At the age of peak risk of drowning (1 to 2 years), it is apparent that the atavistic diving reflex still operates to some degree. Its presence can still be demonstrated in adult human volunteers.[5,6,17] Before consciousness is lost, it may be predicted that, within seconds, the submerged victim manifests bradycardia and shunting of blood from the cutaneous and splanchnic vascular beds to the cerebral and coronary circulations.[5] Blood pressure promptly starts to rise.[6] These reflex changes are independent of baro-receptor and chemo-receptor inputs and depend both on sensory afferents in the trigeminal nerve, and on reflex or voluntary inhibition of the medullary respirator centres.[17] Variation in water temperature above 20°C (the most common situation in the case of real-life drownings)[18] does not influence the brain-protecting diving reflex,[7] but progressively lower temperatures augment it.[19] I have speculated that the brain-sparing effects of cold water[20] may be mediated through an augmented diving reflex as much as through physical chilling of the body core.

It is known that the diving reflex is more intense in the frightened or startled mammal, compared to that observed in an animal which dives or submerges voluntarily.[15] If this is true in humans, a case can be made on physiological grounds against the practice of so-called "drown-proofing" of young infants which is condemned by most paediatric bodies throughout the world.

** These experiments were not as cold-blooded as this text reference suggests. They were undertaken by the Canadian wildlife authorities in an attempt to develop a more humane trap; in the fur trapping of mink, muskrat and beaver, the traditional underwater traps caused the animals much distress.

The breath can be held voluntarily until the breaking point is reached. The period of breathholding[21] (and the onset of the breaking point) is determined both by hypercarbic and hypoxic drives. The influence of high carbon dioxide levels and low oxygen concentrations is mutually synergistic; the breaking point occurs at $PaCO_2$ levels below 55mmHg when there is associated hypoxia and at PaO_2 levels below 100mmHg if the $PaCO_2$ is high.

When the submerged victim reaches the breakpoint, and an inspiration has to be made, tachycardia and arterial hypoxaemia have already developed. Gasping follows; glottal spasm may then occur.[22] Besides gasping, large quantities of water are usually swallowed,[23] and, even before consciousness is lost, vomiting with aspiration of gastric contents is likely to occur.[24]

In older children and adults, the dangerous practice of hyperventilation before underwater endurance games or dives can change the dynamics of the breath-holding breakpoint. If the hypercarbic drive to breathe is lost, unconsciousness from cerebral hypoxia may occur before any breakpoint is reached. Under these circumstances, breathing (with fluid aspiration) may recommence after the submerged person loses consciousness.[25]

Within seconds of the first submerged breath being taken, a phase of secondary apnoea occurs,[22] and is followed by involuntary gasping underwater which may continue for several minutes before final respiratory arrest occurs. Arrhythmias are inevitable in all cases of near-drowning and in the absence of ventilation, lead within minutes to brain death if rescue and resuscitation are not effected. The spleens of drowned victims are relatively bloodless;[10] hypoxia in the submerged victim causes reflex constriction of the splenic vessels.

The duration of hypoxia to cause death is age dependent. After extensive interviews with parents, neighbours and rescuers in the Australian studies (using a "bracket method"), it was suggested that children immersed for three minutes or less are likely to survive; and that the necessarily crude estimates of immersion time for human fatalities lie between three and ten minutes, provided that the immersion occurs in warm water not below 15°C-20°C.[18] Long immersion times (5 to 20 minutes) are still occasionally compatible with survival and the preservation of normal intellect,[26] even in warmer water; and very long immersion times (5 to 40 minutes) are certainly compatible both with survival and with restoration of normal neurological function in children who have been immersed in very cold water (0°C-15°C).[20] In experimental animals, survival times were marginally longer after immersion in salt water.[22]

Consciousness is always lost within three minutes of involuntary submersion. Even in swimming and diving animals, such as minks, consciousness is lost within 2.5 minutes, and the EEG becomes flat within 4.5 minutes.[15] In practice, when the human victim loses consciousness, this is almost always because of cerebral hypoxia. In some boating accidents, in accidents involving falls through ice, and in coldwater accidents involving victims who can swim, immersion hypothermia can occur in the absence of primary drowning asphyxia. Consciousness is lost when the core temperature falls below 34°C.[20]

Inhaled Fluid

The salinity of sea-water varies, but typically sea-water contains 34.48g/kg (3.5%) of dissolved salts, of which 29.54g/kg (2.9%) is sodium chloride. Fresh water contains variable amounts of organic material, dissolved salts, and free and nascent gases.

Most victims who drown, do so in the sea, in chlorinated fresh-water pools, or in bathtubs with variable concentrations of soap. The chemical effects of chlorine and soap in fresh water are currently believed to be of no consequence in the pathophysiology of lung syndromes in survivors, and laboratory studies have shown that the effects of chlorinated fresh water and of simple fresh water on the surface tension of lung surfactant are similar. However, compared with the effects observed after salt-water aspiration, both types of fresh water considerably elevate the minimum surface tension of tracheal and lung surfactants.[22] Sea water, and water with sodium chloride concentrations which are approximately iso-osmolar with plasma, do not denature pulmonary surfactant, but may dilute it or wash it out.

In "wet" drownings, experimental studies with anaesthetised dogs have shown that, even if large amounts of fluid (for example, 22mL/kg) are aspirated as a single event, spontaneous breathing (albeit with a grossly altered ventilatory pattern) will continue.[27] One implication of this is that, in a typical childhood drowning (say, that of a 13-kg, 2-year-old toddler), the amount of inhaled water needed to drown the child would probably exceed 0.3L, in a true "wet" drowning.

Although children drown occasionally in vessels filled with paint, fertiliser, and agricultural, industrial, and domestic chemicals,[23] such occurrences are rare in the broad perspective of accidental immersions.

The Lungs

The "march of events" in the lungs of a drowning victim is summarized as follows:

1. Increased peripheral airway resistance.
2. Variable degrees of laryngeal spasm.
3. Reflex pulmonary vessel vasoconstriction, leading to pulmonary hypertension.
4. Decreased lung compliance.
5. Ventilation-perfusion ratios fall.
6. Fluid shifts occur across the alveolar membrane.
7. Surfactant loss occurs (salt water), or its properties alter(fresh water).
8. Foam and froth production.
9. Anatomical changes in alveolar epithelial cells.

When fluid is inhaled, vagal efferents cause obstruction of the peripheral airways.[28] Fresh water inhalation is particularly effective as a trigger. For this reflex; osmolarity and not volume is the more important trigger. Even when very small amounts of fresh water are inhaled (1mL/kg in experimental animals), aspiration is followed by pulmonary vasoconstriction, with the immediate development of pulmonary hypertension induced by a parasympathetic reflex.[29] With the aspiration of larger volumes of water (for example, 2.5 mL/kg) intrapulmonary reflexes lead to the blood perfusion of non-ventilated areas both in experimental animals,[29] and in humans.[13] This phenomenon (combined with the loss or inactivation of surfactant, and compounded by alveolar collapse) causes a significant reduction in mechanical compliance.[3] In normal lungs, about 5%-18% of blood perfuses non-ventilated areas; within minutes of the aspiration of even small amounts of fresh water this rises to 75%.[27] Clinical studies in humans have shown that, even in near-drowned children who rapidly (within minutes) become virtually "normal" from the clinical point of view, the redistribution of blood perfusion takes at least several days to revert fully to pre-accident levels.

Inhaled water moves across the alveolar epithelium, through a basement membrane, and finally through the endothelial capillary lining into the capillary lumen before haemodilution occurs. This flux causes rapid and severe distortion of pulmonary ultrastructure. Histological examination shows damage both to type 1 and to type 2 epithelial cells (pneumocytes); the latter cells are believed to produce surfactant. Endothelial changes consist of microvesicle formation, swelling, detachment from the basement membrane and disruption of cells.[30] It is probable that this phenomenon is intensified if alcohol and other gastric contents are inhaled.[8]

Denaturisation of surfactant can continue even after an apparently successful initial resuscitation. This phenomenon is known as "secondary drowning", and is potentially fatal if not recognized and treated. Clinically evident secondary drowning occurs in about 5% of survivors,[31] but it is probable that some degree of hyaline-membrane formation occurs in most cases of near-drowning. An outpouring of a proteinaceous exudate which is relatively cell-free occurs and may take several days to resolve.

Mechanical blockage of the bronchial tree is produced by a combination of bronchial spasm, changes in the elastic properties of lung tissue, and an increase in non-elastic resistance caused by physical blockage of the airways by fluid and foam.

The Progress of Hypoxia

The cause of death in an acute drowning episode is irreversible cerebral anoxia.[3] Brain death in the drowning victim may occur before or after cardiac asystole. In about 5% of survivors who persist in a totally "vegetable" state,[26] it may occur without any long-term evidence of coronary hypoxia.

After consciousness is lost, there remains a short period of reversibility of the altered neuronal metabolism, before intracellular hypoxia and acidosis

cause permanent damage.[32] Not all neurones are equally vulnerable, and those associated with more subtle forms of intellectual activity are affected first. In survivors of childhood freshwater immersion accidents, I have observed that skills which require visual-motor co-ordination are particularly affected.[26] The EEG becomes flat during this stage, but is reversible. Oxygen depletion arrests neuronal activity, and it is likely that the depletion of oxygen stores progresses more rapidly, and is more responsible for these early changes than is the effect of the accumulation of potentially lethal quantities of hydrogen ions.[33] It is probable that the order of deleterious influence is hypoxia, acidosis and hypercarbia.

The blood-brain barrier breaks down and both fluid and the macro-molecules pass into parenchyma,[34] functional, if not anatomical dissolution of vascular and cell-wall integrity leads to intracellular and intercellular increases in osmotic pressure.[35] If the patient survives to be resuscitated, oedema develops. It is thought that cerebral oedema caused by ischaemia is harmful only in that it may cause swelling of the hemispheres sufficient to result in mass effect and herniation. There is no evidence to date to indicate that oedema per se increases the extent of neuronal death.[38] In survivors, the speed with which significant cerebral oedema develops remains uncertain, but this phenomenon has been reported in several recent studies as not being a real problem in the first 24 hours after rescue.[37-39]

The final stage is that of permanent cerebral death. In cases of true brain death there is no recovery,[34] although patients supported by mechanical ventilation can exist in a state of true brain death for 12 weeks,[40] or longer.

Survivors And Potential Survivors

A child who is still active when rescued, or who has at least some cardiac activity when he or she is in the emergency-room or casualty department, will be at a finite pathophysiological point along the above-mentioned sequence. Continued improvement in the management of near-drowning, so much a feature of recent years, depends on an understanding of this sequence.

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THE ART OF BREATHING

In Emergencies It Becomes A Science

Albert L Pierce

Different ways of breathing under water can either cause or alleviate problems.

If your mask leaks or is knocked off, you could inhale water. Your basic training in scuba should have included plenty of practice in breathing without a mask while underwater, providing confidence in the ability to breathe comfortably without a mask and through your mouth. Then, if the mask leaks or is lost, you will habitually inhale only by mouth and continually exhale a few bubbles of air from your nose to keep the water from entering.

Long, slow breaths are the most efficient for diving. Whenever you are not inhaling you should be exhaling, breathing all the time. Holding a breath is appropriate only when necessary for the task at hand - photographing a fish, for instance. During such breathholds, avoid a lung over-pressure accident by making sure that you are not moving upward, and that there are no high waves overhead. Such waves can cause sudden variations in water pressure just as if you are bouncing up and down under water. Also, avoid filling your lungs to capacity. The alveoli (microscopically small air sacs in the lungs) can over expand if they are full.

BUOYANCY

While diving, you can fine-control your buoyancy by adjusting the volume of air in your lungs. Relatively full lungs (but, for safety, not filled to capacity) will help you to rise. A low-lung volume will help you to descend. By varying your breathing pattern (but still breathing in and out continuously) you can change your buoyancy by four to five pounds without adding or subtracting weights.

On the surface, a large lung volume will help you to float. Here, you can fill your lungs to capacity without danger. Inhale quickly and fully after each exhalation and make your exhalations short to avoid losing the buoyancy you have gained.

SKIP BREATHING

Some divers pride themselves on their ability to conserve air, and do so by inserting long pauses between inhalations and exhalations, in effect skipping a breath. Holding the breath this way allows dangerous build-ups of carbon dioxide that can cause headaches, drowsiness and confusion. CO_2 can intensify other problems including decompression sickness and nitrogen narcosis. Eventual loss of consciousness could occur.

COUGHING

A coughing diver on the surface may not only be unable to catch a breath, he may also be struggling to stay afloat because he is losing buoyancy each time he coughs out air and gets none back. Get to him quickly. If he is not panicky, make yourself buoyant, then give him support or inflate his buoyancy compensator.

A coughing diver under water is likely to dash madly for the surface, holding his breath. You may be able to stop him by grabbing a leg as he goes by. However,

To signal your buddy to exhale use your hand and blow bubbles.

Purse your lips and blow bubbles when you have no regulator inserted.

tests have shown that there is a tendency for a rescuer to hold his breath as the victim drags him up. Do not let the circumstances make you forget to vent air.

If, in your grab, a fin comes off in your hand, its loss may slow him somewhat. Follow him at a slower pace to make sure he hasn't suffered a lung over-pressure accident.

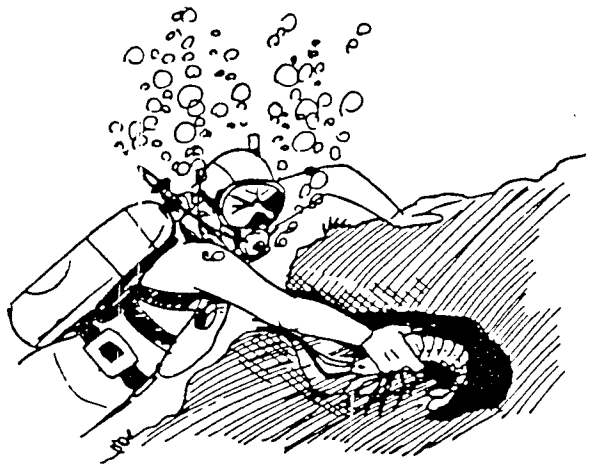
In stopping a panicky diver, do not hold your breath. If you do stop him, do not punch him in the chest to make him exhale. You cannot force a panicky diver to exhale that way. The pressure of such a blow could cause the lung rupture you are trying to prevent. Signal him to exhale using the language of the deaf.

If you have a coughing spell at the surface, try submerging your face. You will gain buoyancy. Your seventeen pound head weighs almost nothing under water. With your mouth aimed down, gravity will help expel the water. Stay near the surface so you can lift your head as soon as the coughing spell is over.

During a dive, water in your regulator could start you coughing. Swallowing may help. But if it does not, cough into your regulator. Do not remove it. The experienced diver will sense the presence of water and inhale slowly and cautiously with the tip of the tongue at the roof of the mouth, thus excluding water droplets from reaching the air passages that could cause coughing or a laryngeal spasm.

EXERTION BREATHING

On land, you can do strenuous exercise and get enough oxygen by heavy breathing. Under water this can be very dangerous. Your regulator may not supply the required large volume of air fast enough.



Most regulators will not supply adequate air for heavy exertion at depth — especially if the tank air pressure is low.

Rapid breathing will not help. If you are able to suck air fast enough through a regulator to double the flow, four times the resistance will be created, and you will use eight times as much oxygen in the attempt. The resulting air hunger will make you breathe faster, a vicious cycle. Most regulators will not supply enough air for even moderately heavy work at 130 feet when the tank is below 300 psi. Some will not give adequate air to support a working diver at only 66 feet. If you are caught in an exertion-breathing cycle, stop working, ascend, and breathe gently. Avoid these problems by taking it easy under water, staying shallow, and surfacing before the tank pressure gets low.

If your buddy starts working and breathing hard, stop him and signal him up. He may want to buddy breathe. That will not help. If your tank pressure is the same and your regulators have similar breathing characteristics he will be no better off. Offering an octopus second stage may make matters worse. The first stage serving both mouth-pieces will be dangerously over-breathed unless you take alternative breaths. An air-hungry diver is not likely to be able to co-ordinate his breathing with yours, and you will both end up without enough air.

A pony bottle or BC breathing would help solve this problem, but prevention is best. Avoid exertion under water, especially at depth with low air pressure.

A diver breathing hard on the surface is probably fighting to stay afloat. He may be using his arms so vigorously that he cannot stop long enough to even reach down for, much less unfasten and drop a weight belt or inflate a BC. So, commanding him to do so may not work.

Asking him to give it to you to hold may present an even more difficult problem. He will not only have to undo his belt, but also hold it out for you while he sinks from inability to use that arm for support. If he is encumbered with goodies, however, he may be glad to give them to you to free his hands. Do not keep what he gives you and drown. Even if it is an expensive camera, drop it if you are not buoyant enough to hold it.

PANTING

Rapid shallow breathing is sometimes called the "hyperpnoea syndrome". Inhalations do not get air as far as the alveoli where oxygen is exchanged for carbon dioxide. Air is simply moved back and forth in the airway "dead spaces". Fresh air does not mix with the stale air deep in the alveoli. So carbon dioxide accumulates and oxygen is depleted. Faster panting simply makes matters worse.

On the surface, the small volumes of air inhaled make a diver less buoyant so he works harder to stay afloat. This exertion increases air hunger and starts another vicious cycle.

The answer is to stop strenuous activity. To do that you will have to make yourself buoyant. Inflate your BC, drop weights or turn onto your back and put your heavy head in the water where it will weigh almost nothing.

Under water, panting is even more dangerous. If a diver cannot be signalled to slow down and breathe deeply, he must be brought back to the surface. Buddy breathing, octopus breathing, or even an auxiliary air supply is not likely to help.

HYPERVENTILATION

You know it is dangerous to hyperventilate (over ventilate the lungs by deep, fast breaths) prior to a long breath-hold dive. You could black out from insufficient oxygen before the carbon dioxide signal to breathe is strong enough to warn you to surface.

Novice scuba divers, however, are liable to hyperventilate under water simply from apprehension

during a dive. The resulting drop in carbon dioxide may cause a tightening of the chest that may be interpreted as shortness of breath. A frightened novice may breathe more vigorously thus starting another vicious cycle. Again, offering another air supply will not help. Long, slow inhalations will rebuild carbon dioxide levels. Signal the novice to slow down and exhale more slowly. If necessary, surface with him.

INHALING WITHOUT EXHALING

A novice diver may generate another vicious cycle of failure to exhale enough. He may inhale fully and exhale only slightly. He may keep this up until he can inhale no more. He feels that he must conserve air under water and holds on to it at all costs. Since he cannot breathe, he may assume that his regulator is not working and try to use someone else's. Obviously, that will not help. With all that air inside, he is also very buoyant and a prime candidate for air embolism. A buoyant diver, struggling to stay down, and venting very few bubbles, should be signalled to exhale.

LUNG RUPTURES

Autopsies have failed to show actual rupture of lung tissue following air embolism deaths. Air may escape through pores in over-expanded alveoli. However, the familiar term, lung rupture is used here. The cause of lung rupture are three fold:

1. All or part of a lung is closed off (or partially closed).
2. That part is fully expanded (Alveoli cannot over-expand until fully expanded).
3. While alveoli pressure builds.

You can avoid a lung rupture by not closing off any airway and by avoiding fully inflated lungs during any period when alveolar pressure may be rising. There is more to this than simply exhaling while ascending.

An airway may be closed off not only by holding the breath. Small airways may also be closed off by unhealthy lungs: tumours, cysts, scars, and excess mucous caused by smoking or a respiratory illness, for instance. Some say you should not dive for up to six weeks after a severe respiratory infection.

You may close off a small airway by exhaling too fast, or too much, or by ascending too fast. The microscopically small airways that connect to the alveoli do not have cartilage rings to keep them open. Excess air flow may cause them to collapse and then trap expanding air behind them. It should be obvious that you should not "blow and go" (exhale completely and swim fast to the surface). Blockages in the lungs are frequently only partial. If you ascend slowly the excess air will have time to escape without over-expanding the alveoli.

Dr George Harper, of Tobermory, Canada, who has done research on this, states that any restriction of air flow, even humming as you rise, may precipitate a lung rupture.

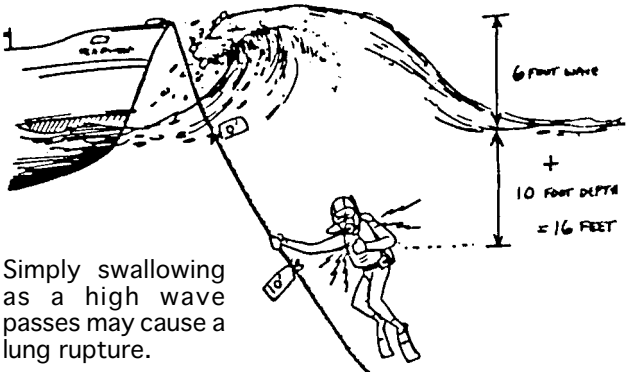
Remember also that the glottis closes as you swallow. Avoid swallowing whenever the water pressure may be dropping.

Since alveoli must be fully expanded before they can over-expand, you can give yourself a margin for error if you never take deep, full breaths.

We all know that the drop in water pressure as we rise can cause a lung rupture if the breath is held, especially at depth. A quick rise from 60 to 50 feet can do it.

Diving when waves are high can be dangerous not only because they toss you around on the surface. The quick passage of a deep wave through overhead while divers were just below the surface is believed to be the cause of at least two lung rupture accidents.

Lungs may rupture even without a drop in water pressure. If air pressure in the lungs is increased, the result can be the same. Inhaling while pressing the purge button on your regulator may be another way to get an air embolism. How we breathe is very important to divers. Habits can help us by eliminating the necessity of remembering. If we habitually vent air as we rise (but not so much that may negate buoyancy), whether swimming, skin diving, or scuba diving, we will automatically, from force of habit, vent air as we rise after losing an underwater air supply.



Simply swallowing as a high wave passes may cause a lung rupture.

Form the habit of inhaling only through the mouth when you are wet and you will avoid getting water up your nose. A habit of constantly exhaling a few bubbles of air through your nose will also keep the water from entering. Practice these habits while swimming.

On scuba, habitually continue deep, slow breathing either in or out. Avoid exertion, stay shallow, and surface before your tank air gets low.

Practice coughing in shallow water with your face submerged, and into a regulator, so you will do it right if caught in deep water.

And above all, avoid lung rupture by habitually keeping your glottis open, your lungs never fully inflated, and by venting air gently any time your alveolar pressure may be rising. If out of air, alternate venting air with attempts to inhale.

Embolism is the leading cause of diver deaths. By applying the techniques of proper breathing, you can assure yourself of continued safe diving.

The author of this article, Albert L Pierce, is currently Chairman of the National YMCA's Scuba Life-Saving Accident Management Program. Much of the material in this article will appear in his forthcoming book Scuba Lifesaving and Self Rescue. The drawings are by Jim Mitchell. Pierce initiated a second career in diving after spending 35 years with the FBI, 32 as an agent.

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DIVING RESEARCH FORGES AHEAD AT UNIVERSITY OF PENNSYLVANIA

The University of Pennsylvania (Penn) has just passed its fortieth year of continuous research investment in the development of modern commercial and military diving technology.

From the beginning Penn has combined basic research, research application, engineering development and operational applications to improve man's capacity for work in the sea. Some of its better known pioneering contributions have included the initiation of practical self-contained diving in the US Navy, Army, and Coast Guard; development of self-contained oxygen and mixed-gas diving apparatus; demonstration of diver lock-out from submerged and underway submarines; accomplishment of midget submersible operations from the deck of a fleet submarine; co-development of the original deck-decompression chamber designed to receive divers under pressure from a submersible chamber; and the initial open sea helium-oxygen saturation dive beyond 200, 300 and 400 feet - the Link-Pennsylvania Project.

Such practical applications were generated by basic research centred at first in primitive facilities in the basement of the University's Medical School. A World War II altitude chamber adapted, by strengthening ports and reversing doors, for use to only about five atmospheres positive pressure, served for many years as the US university headquarters for undersea medical research and communications and for the research training of many of this country's well known naval diving medical officers.

Penn initiated, with the Office of Naval Research, the international series of Underwater Physiology Symposia to stimulate interest in undersea research, and was the headquarters for founding of the Undersea Medical Society.

The present Institute for Environmental Medicine was established in 1968 (15 years ago) as a University-wide organization, on the site of the original laboratory. On completion, it represented the most advanced laboratory system in existence for combining undersea and atmospheric environmental simulation, and has aided the development of naval and civilian laboratories elsewhere.

With a staff of more than 40, it remains among the most versatile, with capacity for basic and applied study of transitions ranging between high and low temperature, positive pressure and high altitude, dry and underwater conditions, and different useful or toxic respiratory atmospheres. Its environmental simulator facilities have been progressively improved since construction and should last indefinitely.

Credit for bringing the Institute and its facilities to its present-day sophisticated state must go to Dr Christian J Lambertsen. An MD pharmacologist, he has exercised his will and sales ability to find sponsors for his projects and to some he is a gadfly, especially in the present day stringencies of government funding. Not only is he a fund raiser but he has gathered together a dedicated team of researchers who are imbued with his enthusiasm to venture into previously unexplored regions. (For a previous story on Lambertsen and his work, see Under Sea Technology for March 1971, page 13).

Predictive Studies

The series of Predictive Studies conducted by the University of Pennsylvania Institute for Environmental Medicine spans the entire period during which most existing commercial diving companies and their modern operations evolved.

Each Predictive Study in the continuing series is a collaborative research program, national in scope, superimposed upon the individual projects of the Institute's investigative staff.

The co-ordinated research in each study is designed to obtain quantitative physiological, chemical and performance information in normal men during graded degrees of the physiological or physical stresses of the undersea working environment. They are therefore focused on determining mental, sensory respiratory, neuro-muscular and other functions which are essential to competent work at depths in the region of probable future operational activity.

The research aim is to measure the degrees of functional competence up to distinct limits of human tolerance, under controlled conditions where harm can be avoided. The information allows the development of dose-effect or stress-effect relationships from light to extreme stress.

Each Predictive Study has required about two calendar years of time for preparation, performance and analysis of results, with an investment usually exceeding forty-investigator years.

Co-operative Approach

A special feature of these predictive research programs is the manner in which multi-disciplinary laboratories, usually from more than one university, have been brought together with offshore industry and federal agencies including Navy, NASA and NIH, to attain a major research and development goal. This planned co-operative approach to large scale laboratory research has allowed rapid application of research findings to work at sea.

The Predictive Studies Programs have not been designed to seek records for pressure exposure. instead, they have emphasized selected measurements, and have intentionally attempted to match the pressure conditions used in prior pressure exposures used by the Institute or by others.

This philosophy of open planning and matching of conditions has provided the ability to correlate results obtained by different laboratories. It is arranged in advance by close international laboratory communication.

In the past several years, other laboratories in Europe and the US have explored extreme pressures in dry chambers, encountering the predicted respiratory stresses of high gas density, and a prominent overlay of undefined hydrostatic pressure effect. Analysis by physiologists at the Institute has indicated the probability that at pressures not greatly in excess of those already attained, the combination of gas density and hydrostatic effects may lead to convulsions and irreversible respiratory failure.

At intermediate pressure, probably at least to the 1600-foot pressure of Predictive Studies IV, fully

competent performance is considered practical from the biomedical standpoint. To improve upon operational capability over the important depth range shallower than this, the present Predictive Study V concentrates on specific investigation of human oxygen tolerance and toxicity. The information obtainable with oxygen at the low pressures of one to three atmospheres is relevant to improvement of diving at all working depths.

Major Studies Findings

Here are selected examples of special steps in Predictive Studies contribution to diving development.

Multiple day saturation with nitrogen to four atmospheres in Predictive Studies I and II produced no harmful effects on bone marrow, respiration, nervous system or physical capability. It was in Predictive Study I that the first observation of bubble formation in the human eye was observed following decompression. The demonstrated absence of nitrogen toxic effect led to open-sea nitrogen saturation and National Oceanic and Atmospheric Administration (NOAA) N_2-O_2 saturation-excursion procedures. These are now coming into active commercial use.

Predictive Study III carried out a detailed physiological comparison of nitrogen, neon, and helium as respiratory inert gases at high pressure. The effects of each on many critical body functions were simultaneously investigated in stepwise compressions in the dry chamber to pressure equivalents of 100, 200, 300, 400, 700, 900 and 1200 feet of sea water. Exposures to nitrogen terminated at 400 feet, while helium and neon comparison continued day-by-day to 1200 feet.

A specific design principle of this Predictive Study was to obtain physiological and other information at several increasing exposure pressures for each gas. This allowed construction of useful "dose-response" or "stress-effect" diagrams, from which practical operating limits could be derived.

The study showed that neither neon or helium had detectable mental or sensory narcotic properties even at 1200 feet. Severe physical work, equivalent to running up the stairs of an 80 floor building, was practical when helium was breathed. The stepwise compression used, permitted progressive adaptation to the high hydrostatic pressure and the subjects were found to be as competent physically and mentally for constructive work while breathing helium at this pressure as on the surface. These findings provided the several bases for the effective and safe use of divers in construction of the Shell "Cognac" platform in approximately 1200 feet of water.

Since neon is about five times more dense than helium, its intentional use as a breathing gas at 1200 feet allowed safe investigation of a pulmonary resistance stress equivalent to what should be expected if helium were breathed at a 5000 foot depth. Without the influences of narcosis or hydrostatic pressure, this extreme respiratory gas density was tolerated for short periods, even in severe physical work.

Predictive Study III also led to the discovery and identification of the new gas lesion disease now called Isobaric Inert Gas Counter-diffusion Sickness. The disease was generated by breathing nitrogen or neon (with appropriate oxygen) while surrounded by helium. It involves gas lesions in the skin, severe dizziness and nausea due to disruption of vestibular function, and a continuous gas embolism in the venous blood.

This represents a third form of gas lesion disease in diving (the three now recognised are decompression sickness, arterial gas embolism due to ruptured lung, and isobaric counter-diffusion sickness). By basic research in animals to determine the cause of counter-diffusion disease, it has been possible to show how to prevent it in offshore operations. The now disease and its principles have now become part of the language and precautions of diving.

The Fourth Predictive Study is best known because it concluded with 1000-foot deeper extension of demonstrated in-water practical work capability, to a pressure equivalent of 1600 feet.

Eleven diving, petroleum and gas companies participated as sponsors in the comprehensive physiologic investigations of rapid compression to helium pressures of 400, 800, and 1200 feet. Excursions from 1200 to 1600 feet allowed study of adaptations to hydrostatic pressure effects upon respiratory nervous system, sensory, blood and older muscle functions.

Decompression from these approximately one hour 400-foot working excursions between 1200 and 1600 feet required only one tenth the time that an equivalent dive from the surface to 400 feet. The principle of excursion from deep helium saturation was also found to facilitate adaptation to high hydrostatic pressure, but the pressure of 1600 feet was predicted to be borderline for such adaptation.

These demonstrations in Predictive Study IV were followed by the open-water test dive to 500 m (about 1600 feet) by Comex and the laboratory 500 m in-water work studies at the Norwegian Underwater Technology Centre (NUTEC).

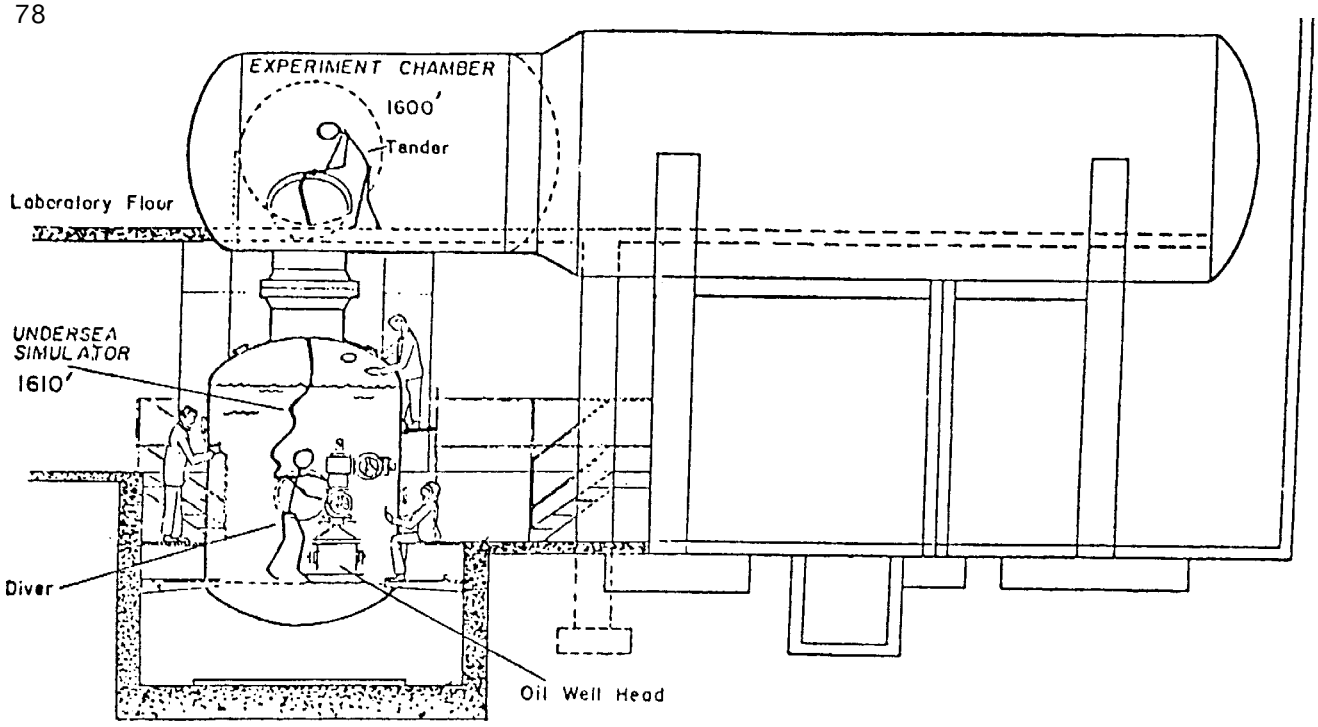
General predictions from the entire series of Predictive studies have emphasized optimism for continuing highly effective use of man throughout the entire range of pressure between the surface and 1000 feet. Providing that remaining bio-medical research and engineering development are accomplished, these investigations indicate that appropriate use of man for work underwater will continue indefinitely to be more work effective and cost effective than exclusively automated systems.

High Pressure Oxygen Tolerance

The Fifth Predictive Study is now in an early stage. It is concerned with tolerance of human organ systems to toxic chemical effects of oxygen, rather than to physical effects of gas density or physiochemical influences of inert gas narcosis and hydrostatic pressure.

The undesirable toxic actions exist with high pressures of oxygen in gas mixtures as well as with pure oxygen. The research program measures the rates at which oxygen poisoning develops in critical organs during continuous exposure to pure oxygen at pressures between one and three atmospheres.

The need to define the characteristics of oxygen poisoning over its full range is that, in spite of its



Environmental Chamber Systems For Physiological and Underwater Work Performance.

toxicity, use of oxygen at increased partial pressures is the chief asset for safety and efficiency in decompression, and in the treatment of decompression sickness. By raising the oxygen concentration at any diving depth, and during decompression, the amount of inert gas (nitrogen or helium) which enters the tissues is reduced. This facilitates decompression and lowers the risk of developing decompression sickness.

When decompression sickness does occur, the primary therapy is the use of oxygen at increased pressure, since pure oxygen or gas mixtures high in oxygen pressure can oxygenate involved tissues and simultaneously speed the resolution of gas bubbles. Unfortunately, the toxicity of oxygen limits both the pressure and length of time for its operational and therapeutic use.

The scope of Oxygen Tolerance Predictive Study V covers a range of critical structures and functions which prior years of investigations in cells and animals have shown to be potential targets for the direct or indirect chemical effects of oxygen.

The Predictive Study V research team involves approximately fifteen MD or PhD investigators capable of measuring chemical and other functions of body systems during the oxygen exposures in the Institute environmental chambers.

Their scientific specialist backgrounds range from neurology and neurophysiology, to ophthalmology, cardiology, pulmonary medicine, otology, liver physiology, biochemistry, endocrine function, and mental performance measurement. An equivalent number of laboratory support staff and a chamber engineer aid in preparing and carrying out each experiment.

This Predictive Study differs from the multiwork, high pressure saturation exposures used on only a few subjects to investigate effects of inert gases, density and hydrostatic pressure. Since oxygen is an active metabolic gas and also rapidly toxic, actual exposure periods rarely last longer than one day.

The exposures must be done in many subjects at several pressures and durations to provide the detailed information required concerning rates of onset and recovery.

The planned end result of the Fifth Study is the construction of predictive oxygen tolerance graphs and tables defining the safe and useful exposures for each organ, such as the ear, or function such as vision. It should provide guidance for deep and shallow operations in the effort to combine safety and effectiveness in diving, decompression and therapy.

It will also provide the baseline information needed for the next Predictive program, namely that of postponing the development of oxygen toxicity to further increase the practical application of high oxygen pressures.

The Oxygen Predictive Study was planned six years ago and was preceded by several years of basic research in animals. One of its current elements, the determination in man of lung tolerance to oxygen was carried out as an initial step. It has resulted in the Pulmonary Oxygen Tolerance Curves and "Unit Pulmonary Oxygen Dose" concept now widely used by industry.

The study was begun in late 1981, but had to be scaled down when general research support in the US deteriorated with the recession. Active efforts are being made by both offshore industry and naval research agencies to provide for its continuation and completion, as a key element in advancement of most forms of diving.

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The address of SEA TECHNOLOGY is Suite 1000, 1117 North 19th Street, Arlington, Virginia 22209, USA.

REHABILITATING THE PARALYSED DIVER

A workshop, held in Alabama, 8-9 March 1984 addressed problems associated with rehabilitating the diver paralysed by decompression sickness/arterial embolism. Six major objectives were:

a) Assessment of the incidence and magnitude of the problem, ie. patients with significant neurological residual from diving accidents warranting extensive rehabilitation.

b) Discussion of the pathophysiology and neuropathy of diving accidents.

c) Discussion of current acute management concepts from first aid through current hyperbaric oxygen therapy.

d) Discussion of a co-ordinated hyperbaric regimen, with Singapore as an example.

e) Discussion of how diving accident patients differ from other rehabilitation patients.

f) Development of guidelines for including appropriate rehabilitation with early management through referral of patients for rehabilitation in their home locality.

program chairman, Dr John Miller, Chairman of the Dept. of Anesthesiology at the University of South Alabama, wrote a preliminary report of the proceedings, which appeared in the April issue of Pressure. Highlights of Dr Miller's report are reproduced below.

On day 1, major discussion centered on the pathophysiology and neuropathy of spinal cord decompression sickness. Although potential flaws were pointed out in the model proposed by Hallenbeck, Elliott and Bove, 1975, as evidenced by the discussion of chronic lesions seen at autopsy following death remote from paralytic decompression sickness injury, no alternative comprehensive model was presented. In a paper by Dr Hayashi et al from Japan, Dr Linaweaver showed a case presentation of upper cervical decompression sickness in a diver who died 15 days post injury. At autopsy, CNS lesions indistinguishable from those proposed by Hallenbeck et al were clearly evidenced.

There was continued discussion about the early loss of CNS autoregulation of the circulation with functional loss of the blood-brain barrier in injured areas in cerebral air embolism, with the concomitant early development of vasogenic cerebral development.

Although this finding contrasts dramatically with the early hours of classic stroke, it corresponds most closely with the findings in CNS trauma. This led to discussion of the value of steroid therapy in both decompression sickness and cerebral arterial embolism. It was concluded that once the injury is established, ie. after about 30-60 minutes, there is probably little value in steroid therapy in these cases, over and above medico-legal considerations. A much stronger case, however, could be made for the judicious use of low molecular weight dextran and mini-dose heparin, early in the management of the diving accident victim. Also, from a number of case presentation throughout the workshop, much clinical benefit appears to be gained from the use of O_2 breathing as early as possible following the injury. Dr How has been able to prevent deterioration during the surface interval between treatments by continuing O_2 at the surface.

The second day was almost entirely devoted to discussion of the principles of rehabilitation, and their particular application to the paralysed diver. Although Dr Clippinger, Co-director of the Rehabilitation Unit at Duke University Medical Center, made a strong case for not treating the diver in any differently from other patients with serious spinal cord injury, the consensus was that such cases should be viewed differently. Clinically, the great majority of diving accident victims ultimately make better recoveries than most other patients, and this recognition by the rehabilitation staff from the outset, can optimize the diver's ultimate recovery. Colonel Jimmy How, the physician in charge of the Singapore Armed Forces hyperbaric unit, discussed the management and rehabilitation of the Chinese diving fishermen of the South China Sea. These patients are handled in a closely co-ordinated manner with impressive sensitivity to their needs medically, socially, and economically. The need for such an approach in the United States was demonstrated by both Dr VanMeter's presentation of severely injured commercial divers, and by Dr Swanberg's personal account of his own major diving accident. Also, Drs VanMeter and Linaweaver pointed out the often deleterious role of the current medico-legal situation in dealing with the commercial sector.

It became clear that progressive involvement with rehabilitation personnel: physical therapist, occupational therapist, clinical psychologist and social worker, early in the patient' management would be of significant benefit to the patient. Complications in the procedure of patient care usually occur in the co-ordination of daily hyperbaric oxygen therapy with the other needs of the rehabilitation program.

Finally, a major problem was discovered in the transfer of a disabled or partially disabled patient from the rehabilitation unit associated with the hyperbaric facility to the patient's home locale. In the truly paraplegic patient, facilities and resources exist for this transfer, but for the patient with significant partial disability (ie. diving accident victims in this category) adequate continuity is by no means assured. Dr Clippinger suggested that this situation could be greatly improved by using the American Spinal Injury Association in Atlanta, as an intermediary in obtaining appropriate continuity of care for the diving accident victim. To this end, Dr Clippinger will be presenting a paper on Diving Accident Management at the next meeting of the Association.

Reprinted by kind permission of the Editor of TRIAGE No. 7, July 1984, the Newsletter of the National Association of Diver Medical Technicians.

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

SURFACE DECOMPRESSION ON OXYGEN

This letter refers to the question of the surface interval when using the USN Surface decompression (oxygen) (SurDO_2) tables. There has been discussion locally as to the course of action to take when the surface interval exceeds 5 minutes (from last water stop to first chamber stop).

Personally, I feel that the 5 minutes is part of the overall profile and, therefore, should be treated like a decompression stop. If the 5 minute period is exceeded, it amounts to omitted decompression.

operationally, it is either forgotten about or an extra oxygen breathing cycle is inserted. Does TRIAGE have any comments on this?

Roy Lewisson
Australia

Reply

The concern for exceeding the five minutes allotted has been around a long time. Until now, nothing has been printed in standard diving texts, and the management (or lack of) has usually been left up to the Diving Supervisor. A call to Dr Thalman, Medical Director at the US Navy's Experimental Diving Unit, for comment, resulted in the news that a new edition of the USN Diving Manual is to be published in 1985, and this very concern will be addressed in Section 8.12.1.1. The procedure as outlined by Dr Thalman is, providing the diver remains asymptomatic he should be committed to USN Treatment Table 5 (Table 1A if oxygen is unavailable). This ensures that recompression will result in at least twice the degree of hyperbaric oxygenation than that of the largest top in the SurDO_2 Tables. Where commercial operations result in chamber stops being longer than prescribed in the current edition of the manual, Dr Thalman cautioned that it might be advisable to switch to Table 6. Should the diver become **symptomatic** during the delay he should be treated as a case of Type II DCS.

Where decompression sickness occurs **during** the chamber stop, the diver should be managed in accordance with the recurrence flow charts of the USN manual.

NADMT Board member David Youngblood, felt that technically, we are looking at a case of delay in ascent as opposed to omitted decompression (terminology that has been a factor in court cases). Dr Youngblood pointed out that more and more physicians and physiologists deplore the whole concept of surface decompression, on the grounds that subclinical decompression sickness is produced, and then treated during the chamber portion of the procedure. It has been well established (by Dr Pilmanis and others) that venous bubbles are produced during SurDO_2 dives, as they have for standard air dives, for that matter. Although the lung is usually an effective "bubble trap", evidence is accumulating that this trap may fail under certain circumstances, permitting the bubbles to enter the arterial circulation. Conditions that may precipitate bubble trap failure include hyperbaric oxygen exposure itself and bubbles trapped in pulmonary capillaries. Each event is thought to produce chemical changes that in turn cause release of bubbles. Although the initial concept of SurDO_2 was to get the diver out of the water as soon as possible, to reduce exposure, hot water suits have largely overcome the concern. Additionally, the competitive element of time, Dr Youngblood added, has been surpassed by a need for quality work on the bottom, as diving tasks have become more sophisticated.

Clearly, Dr Youngblood is not a particular fan of SurD procedures! His recommendations, however, concurred with Dr Thalman's to a large extent. Dr Youngblood felt however, that most delays were the result of poor planning and operational techniques. Therefore, a routine procedure of instituting USN Table 6 might be 'punishing' enough to get the diver team to clean up its act!

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

9TH INTERNATIONAL SYMPOSIUM ON UNDERWATER AND HYPERBARIC PHYSIOLOGY

16-20 September 1986
Portopia Hotel, Kobe, Japan

For further details contact

Undersea Medical Society
9650 Rockville Pike
Bethesda
Maryland, 20814
USA

Telephone: (301) 530 9225

IX INTERNATIONAL CONGRESS ON HYPERBARIC MEDICINE, 1987

The IX International Congress on Hyperbaric Medicine will be held at the Hilton International, Sydney, from 1-4 March 1987.

For further information write to

The Congress Secretariat
PO Box 233
Matraville NSW 2036
Australia

COURSES IN UNDERWATER MEDICINE
AT THE RAN SCHOOL OF UNDERWATER MEDICINE

An Introductory course and an Advanced course will be held later this year.

There will be up to 20 places on each course. The majority of places will be available for civilians. There will be a fee for attending the courses. Last year the fee for the two courses was $536. Those who consider that the fee should be waived for themselves should supply the reasons in writing to the Officer-in-Charge SUM who will pass them on to the Defence Department with his recommendations.

Accommodation is not available at HMAS PENGUIN.

Those interested in attending should write to:-

The Officer-in-Charge
RAN School of Underwater Medicine
HMAS PENGUIN
Balmoral Naval PO NSW 2091