The Editor's Offering

A Happy Christmas and a prosperous New Year to all our readers. With a bit of luck in the mail you will be reading this before Christmas. This may well be the beginning of a new era, a year early, when the Journal arrives early in the month of publication. It is the Editorial intention to aim for posting as near the first of March, June, September and December as possible.

One of the joys of being an editor is when someone writes in congratulating him on the standard of the publication. In 1998 there have been three such letters. Not very many you may say, but it is a positive flood compared with the usual rate of one every four or five years! Full credit must go to those behind the scenes workers, Drs David Davies and John Couper-Smartt, our proof readers, who detect the steady stream of typographical and grammatical errors served up to them. The Editor thanks them for their generous service.

Once again Dr Douglas Walker presents his analysis of the reported Australian diving-related deaths. This time for 1994. Again inexperience and not paying attention to details has taken its toll of Australian divers.

For those who enjoy reading the full stories about these, unfortunately often avoidable, deaths there is a treat in store. DAN South East Asia Pacific, in association with J L Publications, is publishing *Australian Diving Deaths 1972-1993* (248 A4 pages packed with information) which will be available before the end of November for $35 plus postage and packing.

Dr Douglas Walker's reports have been gathered and indexed for causes of death. The second part of the book discusses the breakdown of the reasons why 75 breath-hold divers, 178 scuba divers, 45 hose supplied and 3 rebreather using divers died in these 22 years. The causes of the deaths are clearly displayed, not only the post mortem results but also the causes leading to the initial problem becoming fatal. The Editor must declare his interest in the book. Years ago he decided that the provisional reports should be published as a book, but the process got put aside for other things until John Lippmann took it up. As the Editor's computer held the information it had to be transferred to others for further revision and then it came back for typesetting.

Everyone knows the story of the bloke playing squash who thinks he has been hit behind his ankle as he ruptures his Achilles tendon. Did you know that Achilles tendon rupture is a risk of diving in a dry suit? As the paperboys of the past used to yell, "read all about it, read all about it," with pictures, on page 194.

Of the three papers which form the bulk of this issue one is a left over from Waitangi and the other two were presented in Palau. All three are excellent presentations looking at decompression, decompression illness (or sickness depending on which terminology is favoured), and treatment. It is quite clear that the decompression illness which recreational divers present to hyperbaric units is often more resistant to treatment than occupational divers, whether military or civilian, who have been trained to regard any symptoms after a dive as possibly due to decompression and so worthy of reporting. At least one commercial diving company has a rule that any signs or symptoms after a dive must be given a test of pressure. The stipulated test of pressure is a US Navy table 6. Much better to be recompressed unnecessarily than to miss recompression when it is necessary.

Next year's Annual Scientific Meeting will have both Dr Richard Moon and Dr Alf Brubakk as guest speakers. This issue is a chance for our readers to update their knowledge from the couple's separate papers before the 1999 Annual Scientific Meeting, the theme of which is *Gas Bubble Injury and its Treatment*.

Dr Paul Langton has provided an insight into the endothelium which is more deeply scientific than his presentation at Waitangi, where the title was *A Layman's Guide to the Endothelium*. Cardiologists must live in a world of abbreviations as, even after Dr Langton kindly simplified his original draft so that the Editor could understand it properly, the paper now contains a table of abbreviations, 28 of them and their full meanings, to help those who, like the Editor, have problems remembering which abbreviation means what. That said, the topic is very interesting and much more complicated than it used to be when the Editor was learning enough about homeostasis to pass the anaesthetic fellowship. Well worth reading.

A recent development in the Antipodean diving medical world is the very recent formation of a diving medicine special interest group (SIG) by the Australian and New Zealand College of Anaesthetists (ANZCA). The SIG is an educational body whose first members are fellows of the College. But the aim is for it to become open to all those with an interest in diving medicine. As yet the SIG is but an infant with a constitution but, at the time of writing, there has not yet been a meeting. The Editor and other anaesthetists on the Committee think this is a good development for diving medicine and have joined the SIG. The Committee meeting on 14/11/98 will formulate an official position. There will be more information about the SIG in the next issue of the Journal after the Committee deliberations.

ORIGINAL PAPERS

AUSTRALIAN DIVING-RELATED FATALITIES 1994

Douglas Walker

Key Words
Accidents, deaths.

Summary

The available information includes only one snorkel user as having died during 1994, almost certainly greatly understating the truth. This is indicative of the problem faced in collecting a complete list of such cases, a difficulty less likely to occur with scuba or surface supply diving as the equipment from such fatalities is now required to be sent for checking by the relevant State Police Diving Unit. There were nine (9) identified scuba divers who died and three (3) divers who were using surface supply.

Breath hold (snorkel) fatalities

BH 94/1

The victim was a member of a group of visitors from overseas, all aged over 60. On one of the regular tourist trips to a Barrier Reef cay they had a trip in a glass bottomed boat to view the reef below and then went swimming. After lunch they were offered the use of mask, fins and snorkel and given some basic advice. This man chose to decrease any risk by wearing a life jacket, which made him both buoyant and very easy to observe by the safety watch. The crew included a diving instructor who advised the victim, and others, on the correct manner of water entry (walking backwards when wearing fins). The instructor watched the victim for a time after he entered the water and formed the opinion that he was competent.

The tender from another day-trip boat, as it was collecting its passengers, sent its wash across where the first boat's passengers were swimming. Its crew noticed that one snorkeller seemed strangely unresponsive to the wash and went over to investigate. They found that he was unconscious, floating face down, and pulled him into the tender. They commenced CPR and there was sufficient response for him to be still considered alive when he reached hospital but he died there next day.

The autopsy established that he had suffered an acute myocardial infarction. His medical history, as given by his widow, was minimal. He was described as possibly over weight and suffering from mild hypertension, for which he was taking (unidentified) tablets, but was apparently in good health. It was noted that vomiting made the provision of efficient CPR difficult, a frequent finding where resuscitation is described, as is the finding of rib fractures resulting from heroic efforts at resuscitation.

SNORKELLING AT SURFACE IN A CROWD. SILENT SURFACE DEATH. MILD HYPERTENSION ON THERAPY. WEARING LIFE JACKET, BUT FLOATED FACE DOWN. CPR DELAYED DEATH BUT FRACTURED HIS RIBS. ACUTE MYOCARDIAL INFARCT.

Scuba diver fatalities

SC 94/1

Lack of adequate appropriate experience is often the critical adverse factor in diving accidents and in this incident it was certainly significant. The victim had been trained for two years and made about 20 dives, but this was to be her first surf entry. Her buddy had dived for three years and made thirty dives. He regarded her as being somewhat inexperienced, but capable of deciding whether she could manage the conditions of the proposed dive. This was their second dive using their hired equipment and there had been no problems with it on the previous occasion. This time he omitted to check his companion's equipment before they entered the water, with their fins on, moving sideways and holding onto each other as they penetrated what was described as "moderate surf" and were buffeted by it. They separated when able to start swimming and the buddy reached the calmer water beyond the surf zone before looking back. He saw her 5-10 m away, floundering in the broken water. He had been using his snorkel as he swam out and it must be assumed that she did the same. He saw that she did not have the regulator in her mouth but did not notice whether she was using her snorkel. He swam back to her and gave her his secondary (octopus) regulator because he did not know if her regulator was working, then towed her out to beyond the surf line before they descended together in 3 m of water. He noted that she was breathing quietly at this time, then checked that her regulator was functioning correctly.

They remained in contact as she continued to hold his octopus regulator in her mouth, his hand on her buoyancy compensator (BC). He heard her call out (through the regulator) and saw her camera floating away. He retrieved it and they then both held onto its strap. He had been about to indicate to her that she should change to her own regulator and return to the shore with him. During this retrieval the regulator may have been pulled from her mouth but he saw she was breathing normally. A short time later he saw her face up on the sea bed and thought she would breath easier if face down so he tapped her mask. It was

only then he realised he had observed her exhalation bubbles only once in the past ten minutes and that she was unconscious, one hand holding the regulator in her mouth, the other holding the camera's strap.

He attempted to inflate her BC but failed because it vented as fast as he filled it, the reason for this was sand in the BC oral inflation tube valve which was detected when her equipment was examined. It is assumed that the sand entered the valve at the conclusion of the previous dive and was not washed out after the dive. He was also unable to ditch her weight belt, because her small size caused the BC to cover her belt. Despite inflating his own buoyancy vest he lacked sufficient buoyancy to bring her to the surface (he forgot to ditch his own weights) and towed her back underwater. He received some assistance from another diver in bringing her ashore and up onto the beach. There CPR was commenced and the emergency services attended. She died in hospital two days later without recovering consciousness. No medical cause, other than near-drowning, cerebral anoxic damage and terminal pneumonia, was found at the autopsy. It is not known why she failed to use her own regulator in preference to her buddy's.

TRAINED. SOME EXPERIENCE. FIRST SURF ENTRY. MISJUDGED ABILITY TO MANAGE ROUGH WATER CONDITIONS. VALIANT BUDDY EFFORTS TO ASSIST. SHORT STATURE AFFECTED ACCESSIBILITY TO QUICK RELEASE FOR WEIGHT BELT. SAND MADE BUOYANCY VEST VENT. CONTINUED WITH USE OF BUDDY'S OCTOPUS REGULATOR INSTEAD OF RESUMING USE OF OWN REGULATOR.

SC 94/2

Divers are human and suffer from the same range of problems as do others. This man had been the passenger in a road traffic accident (RTA) where another person was killed and it had a severe effect on him because he had been aware that the driver, a workmate, had drunk too much but had been unable to prevent him from driving. He lost weight and suffered from headaches after the accident but refused to seek treatment. His mother had been unaware of the reason until the police called on him for a statement. He took up scuba diving about 5 months after the accident and progressed to dive master level, liking to dive alone. There was an episode of possible suicide attempt, friends saving him. A further stress was when a girl friend became pregnant (by another) and chose to abort, refusing his offer of marriage. His preparations were careful and successfully executed. He left a note confirming his intentions. No warning signs of his risk of suicide had been observed.

TRAINED. EXPERIENCED. FEELINGS OF GUILT FOR NOT PREVENTING ALCOHOL RELATED FATAL ROAD TRAFFIC ACCIDENT. DEPRESSION. PREVIOUS ATTEMPTED SUICIDE. SOLO DIVER. SUCCESSFUL SUICIDE.

SC 94/3

In this case hubris and an apparently minor engineering inadequacy led to death. He was very experienced, including cave and deep diving, and an instructor in several organisations. He was reputedly obsessive about diving safety and had been enlisting the help of an engineer at work to improve a switch unit to make changing from one tank to another easier when using multiple tanks and differing gas mixtures. He was a teacher of the use of "Nitrox" mixtures and was at this time attending a course on "Trimix" diving. However he had told his girl friend, and others, that he would not be using "Nitrox" for this dive, though he had used it previously without problems.

His two buddies were also well experienced in deep diving. They intended to dive on a wreck which lay at a depth of 50 m. He was wearing a twin cylinder unit, which the others believed to contain air, and also had a small reserve cylinder on his belt. Although he possessed a full face mask (for cave diving) he chose to wear the more usual "eyes-only" mask for this dive. The twin tanks were borrowed but he undoubtedly knew that one of the cylinders contained a "Nitrox" (50% oxygen) gas mix. The hoses were colour tagged but, for some unknown reason, were attached to the three way control valve other than in the conventional order, which may have been a factor in what occurred.

Their dive was uneventful for 17 minutes. The buddies were close to the anchor line, ready to ascend, when the victim was seen, head down, about 3 m above the sea bed. His fins were twitching and regulator out of his mouth. They righted him and tried to replace his regulator, but failed because he was unconscious and not breathing. One buddy inflated the victim's vest and they brought him up to 15 m, where they remained to commence their planned decompression, letting him continue unaccompanied to the surface. They knew that the boat crew would see and recover him and they would avoid serious decompression risks by following this plan. Deep divers are said to be aware that at depth self preservation may take precedence before taking excessive risks for a buddy which would result in them seriously endangering themselves. In this case they believed that he was dead when reached so took him up to the 15 metres depth deco stop before letting him free ascend or attempting to replace his regulator. They were possibly correct in this opinion. When they completed their decompression 45 minutes later they found the emergency services present and in charge of matters.

Examination of the equipment showed that he had evidently used the "Nitrox" mixture rather than air for all or

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and Experience		Dive	Dive	Depth in m		Weights	
		Victim	Buddy	group	purpose	Water	Incident	On	kg
BH 94/1	66	No training Experience not stated	Training and Experience not stated	Group Separation before incident	Recreation	Not stated	Surface	No weights	- -
SC 94/1	39	Trained Some experience	Trained Experienced	Buddy No separation	Recreation	3	Surface	On	9
SC 94/2	23	Trained Experienced	Not applicable	Solo	Suicide	15	15	On	Not stated
SC 94/3	43	Trained Experienced	Trained Experienced+	Group No separation	Recreation	50	47	On	6
SC 94/4	45	Trained No experience	Trained Experienced+	Group Separation before incident	Recreation	36	27	On	10
SC 94/5	46	Trained No experience	Trained Some experience	Buddy Separation before incident	Recreation	4.5	Surface	Buddy ditched	17
SC 94/6	38	Trained No experience	Trained Some Experience	Group No separation	Recreation	7.6	7.6	Buddy ditched	16
SC 94/7	36	No training No experience	Trained Experienced+	Group Separation before incident	Resort dive	9	9	On	5
SC 94/8	43	No training Experienced+	Not applicable	Solo	Recreation	15	Not stated	Off	Not stated
SC 94/9	40	No training No experience	Trained+ Experienced+	Group Separation before incident	Resort dive	4.5	4.5	On	7
H 94/1	34	Trained Experienced	Trained Experienced	Buddy Separation during incident	Work	4.2	4.2	On	Not stated
H 94/2	60	Trained Experienced	Trained Experienced	Buddy Separation before incident	Work	23	23	On	13
H 94/3	55	Training not stated Experienced	Training not stated Experienced	Buddy Separation before incident	Work	12	12	On	Not stated

DIVING RELATED DEATHS IN 1994

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	Comments
Life jacket	Not appliable	Not appliable	Not appliable	Hired	Silent surface death among others. Found floating face down. Cardiac death.
Failed to inflate	Yes	Adequate	Serious fault	Hired	First surf entry. Rough sea and equipment factors. Buddy unable to ditch weights. Asthma history.
Not inflated	Yes	None	“fault”	Own	Depression after fatal RTA. Suicide.
Buddy inflated	Yes	Adequate	Significant fault	Borrowed	Deep dive. Faulty 3 way selector, so mixing of Nitrox with air. Oxygen convulsion.
Not inflated	Yes	Low	Adequate	Hired	6th dive, 1st night dive. Buddy pair separation from dive master “guide”. Went too deep for experience. Complacent planning control.
Inflated	Yes	Low	Adequate	Borrowed	No dives for 18 months after course. Rough. Separation. Inflated vest tight. Overweighted. Cardiac death ?
Not inflated	Yes	None	Some adverse comments	Borrowed	Newly trained. Night dive. Out of air. Panic ascent. Possibly CAGE
Not inflated	Yes	Adequate	Some adverse comments	Dive shop	2nd Resort Dive. Surface separation then solo dive. CAGE.
Not inflated	Yes	Adequate	Adequate	Own	Solo. CAGE. History asthma. Reason for developing CAGE unknown.
Not inflated	Yes	Low	Adequate	Dive shop	Resort Dive. Separation. Possible CAGE.
No vest	Not appliable	Not appliable	Significant fault	Employer	In tunnel with pumps working. Difficult access. Air intake hose melted. CO poisoning.
No vest	Not appliable	Not appliable	Serious fault	Own	Unexplained rapid ascent. Previous and recent myocardial infarction. Cardiac death.
No vest	Not appliable	Not appliable	Adequate	Employer	Cold water. Unexplained loss consciousness near surface. Inadequate air supply.

most of his dive, a fatal error possibly caused by his unorthodox connection of the supply hoses to the three way gas selector. He had removed the "air safe" (nitrox) tag from the bottle so the boat's skipper/dive master was not aware that he did not have air in both his tanks. There was no gas escape from the gas selector block but there was some leakage within it such that gas from the tank not in use was able to enter the mixture being breathed. This may have delayed onset of the oxygen convulsion which led to his drowning.

The dichotomy which can exist between factual knowledge and its correct application is illustrated by the fact that during the dive the other two divers practised their buddy breathing while at depth, despite one having air and the other Trimix in his tank. Incidentally the latter diver had not taken a Trimix course and was using it "because it was in the tank". This despite them being very experienced "deep divers". Had the victim been wearing the full face mask he used when cave diving he probably would not have drowned.

VERY EXPERIENCED, HIGHLY TRAINED DEEP DIVER. FAILED TO DECLARE HAD NITROX 50/50 IN ONE TANK. NON-COMMERCIAL THREE WAY GAS SELECTOR HAD IMPERFECT SEAL BETWEEN GAS SOURCES. NON-STANDARD TANK/HOSE ARRANGEMENT POSSIBLE REASON WHY HE MADE DEEP DIVE USING NITROX. . OXYGEN CONVULSION. ONE BUDDY UNTRAINED IN USE OF TRIMIX BUT USING IT.

SC 94/4

The four members of this family had completed their diving course one week before their live-aboard dive trip on the Barrier Reef. The victim had missed the night dive the others had made because she had been on duty at the time. They showed documentary proof of having successfully completed the course before being accepted on the boat. There were about 23 passengers aboard and a crew of 10, which included 3 with diving instructor qualifications. When they joined the boat the victim had made, at most, only three scuba dives.

All the passengers undertook the two daytime dives on the first day but some declined the opportunity to make a night dive that evening. The victim and her husband were among the 14 who took up the night dive offer, joined by the three instructors. They were told they were to choose their own buddy pairs. This was to avoid experienced divers having the enjoyment of their, paid for, dives spoilt by being paired with inexperienced divers who might require supervision. Possibly a sensible commercial practice, but hardly likely to maximise diver safety. The passengers were told that they could have an instructor accompany them on the night dive so they asked for one. Unfortunately the offer was poorly communicated to the dive master who was detailed to accompany them. He had an experienced diver, a passenger, as his buddy and was himself suffering from a sinus problem although he had had no problems on the other two dives. The dive master apparently thought his job was only to be a guide and he appears to have been unaware of any responsibility for the safety of the pair. They descended after the two experienced divers and when they reached the sea bed found that the first couple had not waited for them to arrive but had swum away. Thinking they had seen a light disappear behind some coral, the pair set off in an attempt to join their guide, but failed to make contact. The victim's buddy said that early in the dive she appeared to become somewhat disorientated and made her way back to the surface. After a discussion she said that she wanted to descend and continue the dive, which they did.

It is probable that she was over weighted, as her buddy reported that she had no trouble descending but had "a buoyancy problem". After a short time they were surprised to discover they were at 18 m although the dive plan was for no descent below 10-12 metres. The dive master described how he saw them deeper than him but was unable to dive down to them because of an inability to equalise his ears. His attempts to attract their attention by shining his torch failed and he apparently never thought to send his buddy to them and did not regard them as being in any danger. The victim had been swimming a little behind and deeper than her husband and now made a rapid ascent. He noticed that she appeared to be "having some trouble breathing and her eyes were glazed" as he began to ascend with her. However "she suddenly dropped away and dropped like a rock to the sea bottom". He described her as having the regulator out of her mouth, which was clenched tight shut at this time. He was unable to prevent her descent to the sea bed, a depth of between 36 and 46 m (statements vary), where he found the regulator was out of her mouth. He reported that she grabbed his regulator but he was able to get her to use her own, then it came out again and he was unable to replace it.

By this time he was running low on air and had to ascend, never thinking to try to ditch her weight belt or inflate her buoyancy vest . Though there was a safety watch on the boat it was a passenger who heard his calls for help after he surfaced. A immediate search was made and was rapidly successful as she had a cyalume safety stick attached to her tank. When her buoyancy vest was inflated she began to lift off the sea bed and ascent became rapid after the weight belt was removed. The contents gauge was checked on the boat and showed a reading of 20 bar. Resuscitation attempts were unavailing although sufficiently vigorous to fracture several ribs.

The cause of death was drowning. Examination of the equipment revealed no faults, although it was noted that 21 lbs (9.5 kg) weight was excessive. She managed to reach the surface from 10-12 m during this dive but failed to do so from 18 m. In all probability the critical factor was the

reduced buoyancy provided by her wet suit at this depth. It is not known whether she wore the same weight belt for previous dives or whose property it was. Although she suffered from Raynaud's disease and had been advised to cut dive times to 50% of that allowed by the Tables, this was not an factor in her death.

Following this tragedy the Company strictly defined the basic requirement divers must have before they were accepted for the deep, wreck or night dives it ran. They now required an advanced diver certification or equivalent or having made over 15 ocean dives before acceptance for such dives. It is not known whether the de facto the policy of pairing inexperienced divers with each other has been addressed.

NEWLY TRAINED. 6th SCUBA DIVE. 1st NIGHT DIVE. PROMISED SUPERVISION FROM DIVE MASTER NOT PROVIDED. INEXPERIENCED DIVERS PAIRED. SEPARATION FROM SUPPOSED DIVE LEADER. DRIFTED INTO DEEP WATER. OVERWEIGHTED. STARTED ASCENT IN POSSIBLE PANIC THEN ABRUPTLY SANK TO SEA BED. REGULATOR OUT OF MOUTH. VALIANT BUDDY ATTEMPT TO ASSIST WITH OCTOPUS REGULATOR THEN USED HER REGULATOR. REGULATOR DROPPED FROM MOUTH. UNABLE TO REPLACE AS CLENCHED MOUTH. FAILED TO DROP WEIGHTS OR INFLATE BUOYANCY VEST. LOW AIR BUT ADEQUATE TO INFLATE VEST. PANIC A FACTOR.

SC 94/5

Neither of these divers was experienced, indeed the victim had not dived during the 18 months since his basic course ended and his buddy had only completed his training 6 months before this dive. Their intention had been to join a charter boat dive but this was cancelled because of the rough sea and poor visibility, so they went to a popular dive location, a reef reached by a jetty from the beach. There was a swell but no white caps, the tide was incoming and water covered the reef. They decided that they could manage the conditions and completed their preparations. They entered the water with inflated buoyancy vests, deflated them and descended to near the sea bed. Initially they swam into the current and remained on the landward, sheltered, side of the reef. When they agreed it was time to start their return swim they were disappointed to find that the tide had changed and they were again faced with an into the current swim. They decided to swim back under the jetty to exit at the nearby boat ramp and when they surfaced to check their position they found they were now 200 m from the jetty, 100 m from the local boat ramp, and 75 m from the shore They inflated their BCDs and decided to make a surface return as the current under water was as strong as that at the surface. The buddy used his regulator but it is not known whether the victim was using his snorkel or regulator for the surface swim.

The buddy became so fatigued during his swim in the rough surface water that he ditched his weight belt. He believed his friend was swimming close behind him and was dismayed to find he was alone when he had recovered from the effort needed to get ashore. A fisherman drove him to a nearby lookout and from there he saw the victim floating face up about 100 m from the shore. He made his way back to the beach and swam out, ditched the weight belt, and managed with difficulty to tow the body back to the rocky shore. The swell was described as being 2 m. He was helped to pull the victim up onto the rocks and then CPR was started, though unavailingly. He noticed that the mask and snorkel were missing, that the vest was inflated and the contents gauge read 50 bar at this time. As the victim was a large man, the buddy very fatigued and sea rough, the buddy was heroic in his efforts.

At the autopsy, conducted with respect for the methods required for a diving-related death, it was noted that there an atheromatous plaque producing a 40% narrowing of the left anterior descending coronary artery. However this was not considered a significant factor in his death. His drowning resulted from their failure to recognise that the sea conditions were too marginal for their ability, probably due to their inexperience. Other factors were that he was over weighted (15 kg) and the borrowed buoyancy vest was too small for him so was constricting when fully inflated (though not when only part inflated at the time of their water entry). The other equipment was hired and without faults.

TRAINED. GROSSLY INEXPERIENCED. NO DIVES SINCE COURSE 18 MONTHS BEFORE. TIDE CHANGED SO RETURN SWIM AGAINST CURRENT. SURFACE RETURN WITH INFLATED BUOYANCY VEST IN ROUGH SEA. SEPARATION. OVERWEIGHTED. FAILED TO DROP WEIGHT BELT. 40% NARROWING OF CORONARY ARTERY. DROWNED. VALIANT RESCUE EFFORT BY BUDDY. BORROWED VEST TIGHT WHEN INFLATED

SC 94/6

Only one of the six divers making this group night dive had any significant experience, though two had made previous night dives. The victim had only completed his basic training course two weeks before this dive, which was on an underwater trail marked by a chain on the sea bed. After about 26 minutes in water 9.5 m deep, they surfaced. By now the victim was low on air although the others had sufficient remaining to dive again. When it was suggested they make another dive, and he was offered the loan of another tank, he was able to continue with them. This borrowed tank was less buoyant than his usual tank and the inflation button on the BCD was different, which was explained to him.

Only four chose to make the second dive and one of them experienced difficulty in equalising his ears early in the dive and surfaced, accompanied by one of the others. After a surface discussion he decided to swim back to shore and the other diver then rejoined the other two. The trio followed the guide chain for a time, then realised that they were following a different route and going away from rather than returning to the entry point so they decided to return to the surface. Their depth was 8 m and when they were at 4 m the victim indicated that he was out of air and started to swim vigorously towards the surface. One of his buddies tried to assist him, holding his equipment and pulling him. He pulled his mask off and let the regulator fall from his mouth during his ascent and at the surface he gasped a few short breaths and said he was out of air. He looked around but failed to use the buddy's offered octopus regulator, and was thrashing about trying to remain at the surface. He appeared to be pressing the deflate button on the buoyancy vest, presumably intending to inflate the vest but he had no air remaining. Then he sank but was quickly retrieved and took a breath, then sank again. He was unconscious when found on the sea bed and the buddy who found him ditched his weight belt and inflated her vest to bring him up. Once at the surface his back pack was ditched and he was towed back to the rocky shore where resuscitation efforts were commenced. Although he lingered till the morning of the fourth day he never regained consciousness.

The equipment was recovered after several days and the tank then contained sea water. No faults were found in the equipment beyond the mention that the buoyancy vest was of a medium size and he was a large man. It was tested by being orally inflated underwater and failed to bring the backpack up so was an unsuitable piece of equipment. However there is no evidence that he ever inflated it. The autopsy was unhelpful because he had already "donated generously" to provide organs for transplant surgery. Although his actions invited CAGE it is unlikely that this occurred, his drowning resulted from his panic response to being over weighted and out of air at the surface, a situation potentially aggravated by his wearing a borrowed tank (heavier than his usual one) and buoyancy vest with a different inflation/deflation button placement to his own unit's arrangement.

He was described as being overweight but not obese. His father regarded him as too unfit to scuba dive so he took care not to let him know of his dive plans. His pre-course medical showed normal blood pressure. He was said to be a smoker and deaf, but inexperience and failure to monitor his remaining air rather than his health were the actual critical factors. It was noted at the autopsy that his heart was enlarged and there was up to 50% atherosclerotic narrowing of some of his coronary vessels, but this was of uncertain significance in this fatality.

RECENTLY TRAINED. VERY LITTLE EXPERIENCE. 1st NIGHT DIVE. OUT-OF-AIR ASCENT THEN LACK OF BUOYANCY AT SURFACE. BORROWED TANK LESS BUOYANT. DIFFERENT BUTTON CONTROLS FOR (SMALL) VEST. FAILED TO DITCH WEIGHTS. NO AIR REMAINING TO INFLATE BUOYANCY VEST. ADVERSE HEALTH FACTORS. DEAF. SMOKER. OVERWEIGHTED. PANIC. VALIANT BUDDIES. DELAYED DROWNING DEATH.

SC 94/7

Two overseas visitors, one of whom did not speak English, decided to see the Barrier Reef and joined a day trip to one of the reefs. There were 100 passengers aboard and they were offered the chance to snorkel or, for an extra fee, make a scuba dive supervised by one of the two instructors there to provide this service. During the trip out a talk was given to the passengers about scuba diving and those showing interest were identified, initially only 7, while 30-40 chose the option of snorkelling. A large school group was aboard and some the girls later decided to make a scuba dive. The school authorities had circulated the parents in advance of this special excursion and none had given permission for scuba diving or supplied them with money for such an option but this did not impede the vessel's operators or induce a response from the teachers supposedly responsible for the safety of their charges. However, events transpired to prevent their diving. The water was described as being sufficiently cold for all who entered the water to be provided with wet suits. There was also reportedly some current and waves.

The first "resort dive" passed without problems and all those involved, who included the two friends, decided to repeat the adventure that afternoon after lunch. The dive groups were unchanged, the victim and her friend with two others led by one instructor, the other three with the other instructor. They were under-weighted but their instructor assisted one to descend after dumping remaining air from her buoyancy vest, then returned to the surface to assist the other. There was no air in her vest so he pulled her down to 1.5 m but she found she was unable to equalise her ears, so rather than abort the dive the instructor inflated her buoyancy vest and told her to follow them, swimming at the surface. After about 5 minutes the one who had descended noticed the absence of her friend and started to ascend to look for her. The instructor realised he could not see anyone at the surface so ascended rapidly and reached the surface first. There he reassured the victim's friend and then called to the vessel's lookout to inquire whether he had seen anything. Then three children, who were snorkelling nearby, said they had seen a woman surface, that she had looked worried and they had later seen her lying on the sea bed below them with no bubbles coming from her. The instructor promptly dived where they indicated and found her, ditched her weight belt and inflated her buoyancy vest and brought her up. Resuscitation attempts were commenced as soon as she was pulled into the dinghy sent

from the vessel. These efforts ensured that she was still living when transported by helicopter to hospital but she died there next day without ever regaining consciousness, the result of cerebral anoxic damage and terminal pneumonia.

The delay of 9 weeks which occurred between the incident and examination of the equipment probably allowed the development of some corrosion changes and damage to an O-ring with some loss of air. However there was no suggestion that equipment factors caused this fatality. The autopsy, in addition to the noted changes, disclosed the presence of massive surgical emphysema of the mediastinum which extended into the retroperitoneal space, and air was present in the subarachnoid space. Aspiration of vomit had occurred into the lungs. The strenuous resuscitation efforts and delay before death occurred make these findings of uncertain relevance in understanding the scenario of this incident. The most likely is that she believed she knew how to scuba dive and decided to try to join the others underwater and succeeded in descending, then panicked and ascended holding her breath. She had retained her weight belt and not inflated her buoyancy vest and possibly suffered a cerebral arterial gas embolism, the reason for her behaviour as witnessed by the children, then sank. Her lack of understanding of English would have resulted in her being totally uninstructed in how to scuba dive safely and separation would have been anxiety producing. The survivor told how their instructor told them before the second dive that if they did not wish to go as deep as he was intending to go they need not follow him. An extraordinary failure of responsibility for the safety of those in his charge. It is tragic that she discovered how to equalise her ears while alone and was thereby able to make her solo dive.

2nd RESORT DIVE THAT DAY. INSTRUCTOR WITH 4 "PUPILS". UNDER WEIGHTED. EAR EQUALISATION PROBLEM. SO LEFT AT SURFACE WITH INFLATED BUOYANCY VEST. MANAGED TO DEFLATE VEST AND DESCEND ALONE. PROBABLE RAPID PANIC ASCENT CAGE. FOUND ON SEA BED. WEIGHT BELT ON. VEST UNINFLATED. DELAYED DROWNING DEATH. INADEQUATE INSTRUCTOR AWARENESS OF NEED FOR ADEQUATE SUPERVISION AT ALL TIMES. ORGANISATION WILLING TO ACCEPT CHILDREN FOR RESORT DIVE WITHOUT PARENTAL PERMISSION. LANGUAGE PROBLEM SO VICTIM WAS PROBABLY TOTALLY UNINSTRUCTED.

SC 94/8

This man, although an active asthmatic and untrained, had been diving frequently for twenty years, usually alone. He reportedly claimed he felt better after a dive. There are no details of his asthma's severity or his management routine. There were four divers making this boat dive but they dived as a trio group while he went solo. When they returned to the boat after 30 minutes they saw him floating face up about 30 m from the boat, so one of them swam over to him to check. He found the victim was unresponsive, mask in place, weight belt missing, regulator out of his mouth and water covering his mouth. He attempted EAR while towing him back to the boat where the others were able to commence CPR, though no response was obtained. The contents gauge read 110 bar.

The autopsy was preceded by both X-Ray and CT scans and was itself performed with awareness of diving medicine problems. Clear evidence was found of CAGE and the degree of coronary atheroma noted was considered to be insufficient to cause him symptoms. Neither was there evidence of active asthma. As he was an experienced diver and had sufficient remaining air, the reason for this incident remains unknown. He was reported to have the habit of using his "Ventolin" (salbutamol) inhaler before he dived and although none of his companions saw him use it on this occasion, an inhaler was found in the pocket of his buoyancy vest.

UNTRAINED. EXPERIENCED. 20 YEARS REGULAR DIVING. ACTIVE ASTHMA OF UNKNOWN SEVERITY. NO DETAILS OF ASTHMA MEDICATION USED. ADEQUATE REMAINING AIR. DITCHED WEIGHT BELT. BUOYANCY VEST UNINFLATED. FLOATED FACE UP WITH SUBMERGED FACE. CAGE.

SC 94/9

There were 21 passengers making the trip to the Barrier Reef. During the outward trip the diving instructor told them that, in addition to snorkelling, they would have the opportunity to scuba dive on the reef once they arrived there. Medical history forms were distributed to those interested. After the vessel was moored he gave a 5-10 minute talk on how to enter the water, equalise the ears, clear the mask and purge the regulator, before the first four passengers entered the water. He took them down the shot line one by one, to ensure they managed equalisation of their ears. One of the group had made a single previous "resort experience" dive and the victim (and possibly her husband also) had made two and was keen to make more. The water was described by the people who went snorkelling as being cold, but none of the scuba group mentioned this.

The victim and her husband were excessively buoyant, the former floating up to the surface several times before the instructor handed each an extra weight. Her husband placed his in the pocket of his buoyancy vest while she apparently kept hers in her hand. The instructor led, maintaining hand contact with the other two of his group, with the victim and her husband in the rear, supposedly keeping close to him. When they reached an open area where

the depth increased from 4 m to 7 m the instructor stopped and checked their contents gauges. He noted that the victim's gauge read 70 bar and her husband's read 50-60 bar. He decided it was time to bring them back to the surface, then noticed that one diver was now missing. The dive had lasted 15 minutes. There had been no indication of anyone having a problem. He brought the three who remained to the surface and then saw the skipper coming towards them in the vessel's dinghy. He had seen a solo diver surface and although there was no signal or sign that assistance was needed he decided to go and check the diver personally. This diver had been observed to remove the regulator from (her) mouth in a calm and "professional" way before descending again. The surface was sufficiently rough to prevent them determining whether any bubbles were coming to the surface when they looked.

After an unsuccessful search, till he ran out of air, the instructor returned to the yacht with the others and there he obtained a fresh tank and asked one of the crew to dive with him. Although not formally trained, the instructor had given this man sufficient instruction in the past to believe he would dive safely in this emergency situation. One of her fins was noticed, then her body close by, about 15 m from her last known position, depth 9-10 m. The instructor brought her to the surface by inflating her buoyancy vest. He ditched her weight belt at the surface. Resuscitation was commenced in the water and maintained until the helicopter arrived with a medical team who took over management. She died in hospital early next day from the cerebral anoxic damage she had suffered.

When examined later no fault was found with the equipment, and the contents gauge then showed 28 bar although the reading was higher before the instructor inflated her buoyancy vest. It is unknown why she left the group and made a solo ascent as she had more air than her husband when the signal to ascend was about to be given. Autopsy disclosed that there were fractured ribs, and a large paravertebral haematoma extended from the retropharyngeal level to behind the upper part of the stomach, involving the oesophagus and bronchi in the upper mediastinum. Although the pathologist thought that this represented barotrauma, resuscitation trauma is a more likely explanation. However, a cerebral arterial gas embolism may indeed have occurred and would explain her observed behaviour after she surfaced.

RESORT DIVE (3rd). SEPARATION AND SOLO ASCENT WITHOUT WARNING AS GROUP ASCENT ABOUT TO COMMENCE. SEEN TO SURFACE AND REMOVE REGULATOR FROM MOUTH THEN RESUBMERGE. APPEARED CALM. RESUSCITATION RELATED FRACTURED RIBS AND PARAVERTEBRAL HAEMATOMA. POSSIBLE CAGE. UNEXPLAINED REASON FOR HER ACTIONS. ADEQUATE BUT LOW AIR FOR ASCENT. FAILED TO DITCH WEIGHTS OR INFLATE BUOYANCY VEST AT SURFACE. LOST ONE FIN TERMINALLY. CLINICALLY CAGE.

Surface supplied diving fatalities

H 94/1

Commercial diving is frequently undertaken in less than ideal situations and with the unspoken understanding that tasks must be completed as inexpensively and rapidly as possible if there is to be any future work from the client. Undoubtedly such factors played a significant part in this tragedy. The job was an annual cleaning contract, at a power station, to clear growths and deposits from the guides in which a stop-door ran to cut off the water intake tunnel to provide water free access to clean the intake screens. They were also to replace the worn sacrificial anodes which protected the metal from electrolytic destruction in the presence of sea water. Access was difficult and made no easier by power station economies, if such were the reason for the staff reduction which resulted in the non-provision of a stand-by on-site crane, which would be required if a diver became injured and required lifting up from the tunnel to ground level.

Ladder access was down a shaft to a ledge at about water level, from which the intake tunnels could be entered. The industrial/commercial imperative was that the power station had to maintain operation of the intake to at least half of its intake pumps if electricity was to continue to be generated and this necessitated care on the part of divers to ensure they avoid straying across the entry to the in-operation tunnel.

The compressor was near the top of the shaft and the supply hoses for the two divers were measured out to allow just sufficient for them to reach the work area but no further, then tied to a railing at the top of the shaft. This made it difficult to claim that the third member of the team was tending the hoses while he minded the air compressor. One diver used a water gun to remove marine growths, a tiring and heavy task which he shared with his colleague, who was meantime replacing the anodes. This task involved him removing the old ones and returning to the bottom of the shaft to obtain fresh ones. He was, in fact, a fellow diver most of the time rather than a safety backup for the diver with the gun, which was very noisy.

This second diver noticed that he was becoming ill, feeling breathless and vertiginous. He returned to the shaft to tell the supervising diver his condition, unaware of the reason for his symptoms. His buddy had been close to him and still using the water gun when he decided he needed to get back to the surface in the shaft. The supervisor told him to return and bring out his buddy, which he attempted but he was unable to find him or to pull the hose back, and returned to report this to the supervisor. The supervisor now noticed that a hole had melted in the air intake hose where it

had come in contact with the hot compressor and that this was allowing exhaust fumes to enter. He quickly changed over to the emergency cylinder air supply, but this air was only available to the diver after the contaminated air in the hoses was cleared by usage. He jumped into the water and took the regulator from the ill diver and entered the intake tunnel. The victim's hose was found to be entering the operational tunnel, which was next to the one in which they had been working, and he also was unable to pull the victim out by pulling on the hose so returned to the surface and hit the emergency stop button, to stop a rotating screen, and yelled to workmen to stop the intake pump. He then re-entered the tunnel and retrieved the victim's body, having first to ditch his weight belt. It is assumed that the victim was making his way back when rendered weak or semi unconscious by the carbon monoxide and the intake flow in the working tunnel sucked him into it.

When the victim was brought out to the open water of the shaft he was placed on the ledge there, which was covered by a shallow depth of water, and resuscitation attempts were then commenced. By this time there were others present and assisting. In recognition of the difficulties of treating him on this ledge a loop of rope was placed under his shoulders and he was hauled to the surface. It was in just such a situation that a cradle lifted by a crane would have been useful. There was no response to their efforts. The surviving diver managed to climb the ladder back to the surface, a remarkable feat for someone as effected by carbon monoxide poisoning as he was. He was offered no treatment for his carbon monoxide poisoning until he reached hospital

The autopsy confirmed that carbon monoxide was the cause of loss of consciousness and drowning. It is possible that he would have survived if he had been wearing a full face mask, but he had a habit of vomiting when he began any dive and found it very inconvenient if wearing such equipment. The buddy was wearing such a helmet and disliked it intensely because it was too small for him. There was a helmet among the equipment they carried but it was not used. There was also a get-home bottle, similarly not in use. Their failure to wear a harness to which their air hoses could be attached was similarly a breach of the regulations.

Examination of the equipment showed that the compressor functioned correctly, although the weather conditions of the day may have caused exhaust fumes to remain around it. The melted intake pipe was of plastic, not a contravention of regulations though inadvisable. Investigation by work safety officers revealed a multitude of failures to conform to regulations, ranging from the omission of written instructions for the management of the work and absence of specific training to act as a diving supervisor to the fact that none of those involved was trained beyond the level of basic scuba diver. The air hoses were not as specified for diving air supply, no harness was worn to retain the air hoses, no get-home bottles were worn, there was no retainer strap on the victim's regulator, and no safety lines were worn. Although there was a get-home air bottle in the equipment they brought, it could not have been attached to the equipment they were wearing. The buddy described the full face mask "helmet" as extremely uncomfortable and said it had excessive dead space, allowing carbon dioxide build up. Similarly the unwillingness or inability of the engineers at the generating station to stop pumps while divers were working in the tunnels was inexcusable from a safety viewpoint, and the failure to have a crane with a cradle made recovery of the victim more difficult. All these factors were capable of adversely effecting diver safety but were not critical in the genesis of this fatality, which was entirely due to exhaust fumes entering through the air intake pipe after it melted. This accident could very easily have been a double fatality.

PART-TIME COMMERCIAL HOSE SUPPLY DIVERS. ONLY MINIMAL SCUBA CERTIFICATION. ADVERSE COMMENTS ON WORK MANAGEMENT AND EQUIPMENT USED. HOLE MELTED IN AIR INTAKE PIPE. ALLOWED ENTRY OF EXHAUST FUMES. INTAKE PUMP ALLOWED TO WORK WHILE DIVERS POTENTIALLY NEAR. VICTIM REFUSED FULL FACE MASK AS OFTEN VOMITED WHILE DIVING. CARBON MONOXIDE POISONING. NEARLY DOUBLE TRAGEDY.

H 94/2

This fatality occurred during an attempt to salvage a fishing vessel whose wreck had been purchased in hope of it being raised and resold at a profit. The first task, to locate the wreck, was successfully accomplished, then the clutter of nets and other gear had been cut away, and now the two divers were in process of placing float bags within the wreck. There was one break in the diving while curious sharks were allowed to prowl and then leave. The dive platform was a fishing boat owned by the man who had purchased the wreck and its two crewmen were acting as dive tenders to the two divers while the skipper maintained a radio and visual check to ensure no boats came near.

The victim was on the A-frame of the wreck, detaching the lift bags as they were sent down and handing them to the other diver, who was below him and in the wreck. After an hour his tender noted that his hose went taut, then slack, and the diver was seen to break the surface near the boat. He looked pale and was too unwell to attempt to climb the ladder back into the boat. He was unconscious when pulled aboard. The buddy was given a 4-pulls command to return but chose to take a routine 12 minutes decompression stop rather than surface immediately. As he alone knew anything about resuscitation this was not started until he came aboard and was unavailing. The autopsy revealed a serious degree of coronary artery disease and this disease is assumed to be the reason he ascended hurriedly

and then died. Although the equipment was in poor condition it was found to function correctly.

SURFACE SUPPLY DIVER. SUDDEN ASCENT. CORONARY ARTERY DISEASE. CARDIAC DEATH.

H 94/3

This also was a salvage attempt, on a yacht sunk in shallow water and now full of sand. It had been purchased by an experienced professional diver (A) who had been involved in its crew's rescue when it had hit the sand bar. He had with him two friends, one of whom (B) also had a financial interest in the outcome of the work in return for supplying advice on use of flotation devices. He had borrowed a hookah unit from another friend. They were on a yacht which they moored a short distance from the wreck in order to avoid any risk of damaging the wreck when they succeed in raising it, and for this reason brought two inflatables with them from which to work on the salvage task.

They placed the compressor in one inflatable, their two water pumps in the other. These pumps were used to operate a venturi tube suction hose sand dredge. The compressor had two outlets, one to supply a diver and the other to inflate the inner tubes and 25 litre plastic drums after they had been placed inside the hull of the sunken yacht. All three were divers and while B used the hose supply while clearing sand from the interior of the wreck another (C), using scuba, was placing and inflating the inner tubes and plastic drums in the wreck. The compressor could not supply sufficient air for both the diver and the inflation. The hose supplied diver (B) complained of feeling short of air, so the scuba diver ceased his inflating. Being unable to continue useful work and feeling very cold, C surfaced. A had been in the inflatable with the compressor and took the regulator from B when he surfaced. A then descended to continue clearing out the sand. Both he and B, the first diver, hand-held the regulator rather than securing the hose to themselves. There was a history of equipment problems on six previous occasions (no details are available) but none are reported on this day.

C was about to remove his scuba equipment when he heard a shout from B who had boarded one of the inflatables after handing over his hose supplied regulator to A. C immediately made his way through the array of hoses coming from the inflatables, which were tied together, and looked around. Seeing nobody at the surface he followed shouted directions and looked down. He saw the air hose and regulator lying on the sea floor and later saw A, the victim, sitting on the sea bed facing towards him with a fixed stare, his dentures hanging out of his mouth. He dived, ditched A's weight belt, and brought him to the surface. Although he was not breathing when he was reached he began to do so when his rescuer put his own regulator in A's mouth. During the tow back to the boat he could be heard taking deep, long, noisy breaths. On occasion water covered A's face despite the rescuer's best efforts. They were carried by a current towards the inflatable, which B had managed, with difficulty, to start (at one time a hose became wrapped round the propeller). C failed in his attempt to catch a rope from the boat but fortunately succeeded in catching hold of a scupper and was helped aboard by B. The victim made no attempt to assist and indeed became submerged at one stage. He failed to respond to their CPR efforts.

In his account of what occurred C described seeing the victim ascend close to the surface then remove the regulator from his mouth and sink. It is probable that he was no longer in control of his actions and allowed it to fall away as he sank. He said that they touched fingers, then A drifted down and away. No reason can be offered for the victim acting as he did, for he was an experienced diver. There was no fault with the air supply and the autopsy failed to define a certain cause of death other than drowning. Although CAGE would be a possibility there is no reason why such should occur in this dive, so the finding of coronary atheroma with a maximum narrowing of 50% is taken to indicate the possibility that he suffered a fatal arrhythmia or other cardiac event not identified. There was a suggestion that he may have mentioned having suffered a chest pain recently, but this is unsourced hearsay. He had mentioned to his friends that he had back pain due to a fall, and had bowel cancer. No evidence of any bowel disease was found at the autopsy.

EXPERIENCED COMMERCIAL DIVER. SHALLOW SURFACE SUPPLY DIVE. REGULATOR HELD IN MOUTH BUT HOSE NOT SECURED TO BODY. NEAR SURFACE LET REGULATOR FALL. UNCONSCIOUS. BREATHED FROM REGULATOR WHEN TAKEN BACK TO SURFACE. POSSIBLE CARDIAC ARRHYTHMIA. COLD WATER A POSSIBLE FACTOR. LOOSE DENTURES.

Discussion

There was only one fatality identified in a swimmer using a mask and snorkel, almost certainly an artifact of the difficulty experienced in identifying such fatalities other than where they occur during a commercial operation such as those taking tourists for day trips to the Barrier Reef. This man was apparently reasonably healthy, although a little over weight and having mild hypertension. There is no practical way in which his risk of suffering this fatal heart attack could have been identified. He died quietly among a group of people and while a general supervisory watch was being maintained. This type of fatality will inevitably occur again from time to time and is an inescapable risk.

Examination of the data concerning the factors which are potentially remediable shows that four scuba divers were

grossly inexperienced (SC 94/1, 94/4, 94/5, 94/6) and two (SC 94/7, 94/9) were supposedly under the direct supervision of an instructor while making a "resort dive". Three divers had had no training. Four trained divers were experiencing environmental factors they had never previously experienced, a surf entry (SC 94/1), a night dive (SC 94/4 and 94/6) and rough water (SC 94/5, who had not dived during the 18 months since completion of the basic training course). In one case (SC 94/4) there was a compounding of adverse factors, pairing of two inexperienced divers on their first night dive, separation from their supposed guide, and greater depth than ever before. The suicide was planned and carried out with such care that nobody could have influenced the course of events.

Although the action of the buddy in case SC 94/1 in assisting his friend in the surf zone was timely and correct, it was unwise for them to have delayed her use of her own regulator after reaching calmer water, but the need to recover the camera explains this matter. No reason is known for the victim failing to use her own regulator after leaving the surf zone but possibly this resulted from a fear of letting go an assured air supply and having to clear water from her own regulator before using it. She may well have been "spooked" by her first experience of really turbulent water. The rescue was seriously compromised by the buoyancy vest and weight belt problems. This illustrates the importance of checking the functioning of all one's equipment before entering the water. It is a wise precept to accept a maximum of one problem at any one time during a dive. If this cannot be resolved the dive should be aborted. If this rule had been followed during this dive the victim would either have resumed the use of her regulator or they would have commenced their return to the beach with less delay and (probably) there would have been no incident to report.

It is difficult to identify the reason for the death of the experienced diver in case SC 94/8, and his habit of solo diving removed the possibility of a witness being able to assist with details. In case SC 94/3 there is some doubt concerning the truth of what actually occurred as the victim had reportedly deliberately misrepresented the gas mix he intended to use and at least one of his buddies was using "trimix" merely because it happened to be in the tank he was using. The significance of the three way gas selector block in this fatality is the attention it draws to the narrow range of safety which is present when using non-air breathing gases, where even an apparently minor fault in a single piece of the diver's equipment can exact a terrible price. To this fault was married the mistake of setting up a non-conventional arrangement of the cylinders and their hoses. Where every procedure must be followed absolutely correctly, and "over learning" should be the minimal standard of achievement, it was folly to introduce such changes.

Concerning the finding of an asthma history in two instances (SC 94/1, 94/8), there is no evidence to implicate this as a factor in either, although in the absence of explanation for what occurred in case SC 94/8 there is a temptation to ascribe guilt by association.

Of the four cases where CAGE was the apparent critical factor (SC 94/6,94/7,94/8,94/9), two occurred during a "resort dive" while supposedly under the direct supervision of an instructor. This shows the need for awareness of the high level of responsibility devolving on those who take untrained persons scuba diving. These two incidents were not due to any conduct by the instructor which was unreasonable, although retrospective analysis inevitably shows that matters could have been managed differently. In case SC 94/6 the victim was left at the surface with an inflated buoyancy vest and told to swim above the underwater group but deflated the vest and made a solo dive. This was not an action which anyone would anticipate. In the second case the victim was known to have adequate air shortly before the group was to ascend and had shown no signs of being other than at ease. It is not possible to watch everyone in a group at the same time, even if there are only four to check, and unexpected actions by others will inevitably beat even the best of observers on occasion. It must be ever foremost in the minds of those responsible for the safety of others underwater that there is often only a narrow margin between a good dive and a disastrous one.

This year there were three surface supply (hookah) diver fatalities. The carbon monoxide risk is well documented from previous cases but is still liable to occur unless the intake is at sufficient height to avoid any possibility of an entry of fumes from the compressor's exhaust, and the hose remains intact. Although the commercial work in case H 94/1 was being conducted by a group of divers without commercial training and using sub-optimal equipment, it was by sheer misadventure that this tragedy occurred. Comment here should also be directed towards the responsibility of the client to provide a safe workplace for divers. In case H 94/2 the victim was probably unaware of his cardiac condition and certainly nobody else suspected he had anything wrong. Although a cardiac cause is suspected this cannot be blamed with complete certainty. Similarly in case H 94/3 cardiac arrhythmia, possibly influenced by hypothermia, may be invoked as the reason for his observed behaviour, though never proven.

In brief, diving can be fatal, and failure to follow the generally accepted and somewhat restrictive advice on safe diving protocols is an adverse factor in many dives which end fatally. Inexperience is, not unexpectedly, a significant adverse element in the safety equation and should be taken into account in the planning of all dives.

Acknowledgments

This investigation would not be possible without the understanding and support of the Law, Justice or Attorney General's Department in each State, the Coroners and police when they are approached for assistance.

Project Stickybeak

Readers are asked to assist this safety project by contacting the author with information, however tenuous, of serious or fatal incidents involving persons using a snorkel, scuba, hose supply or any form or rebreather apparatus. All communications are treated as being medically confidential. The information is essential if such incidents are to be identified.

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ACHILLES TENDON RUPTURE AS A DIVING INJURY

Jim Marwood

Key Words

Accidents, case report, first aid, injury, treatment.

Abstract

A case of ruptured Achilles Tendon, occurring during "giant stride" entry, is described with discussion of cause and symptoms, and notes on diagnosis and treatment.

Case report

As the diver stepped off the left foot, to make a "giant stride" entry, he felt a blow on the back of the left ankle. He began finning, but the ankle felt powerless. When, after a few minutes, there was no improvement he decided to surface and return to the boat.

The dive boat was an open "shark cat" type with a side-entry port over a low step. Conditions were calm, but a low swell caused the boat to roll. The diver recalled that as he strode off, his left heel was unsupported, being over the back of the step, and he was unbalanced by the motion of the boat. He believed he had been struck by a falling plank or dive-weight, but this the boat handler strenuously denied.

At 65 the diver was well over the usual age of diving patients, but apart from age he had no factors pre-disposing to injury. He was reasonably fit and accustomed to manual work. He had logged 55 dives in the preceding twelve months, with some 35 giant stride entries loaded, as he was on this occasion, with cold-water gear and heavy photographic equipment.

On return to shore removal of the left drysuit boot was painful. The diver could walk with a limp, but extreme pain on ankle flexion prevented walking on soft sand. There was a little posterior swelling but no bruising. He had full active ankle movement but was unable to rise up on the left forefoot.

Two days later, as weakness persisted, the diver sought medical advice and with the diagnosis of ruptured Achilles tendon (AT) was referred to an orthopaedic surgeon. Open repair was carried out, followed by six weeks in a non-weight bearing below-knee cast. The patient was impatient about mobilisation and this stretched the repair. The tendon healed with residual muscle weakness. At the time of writing, a year later, he can just support his weight on the left forefoot.

Discussion

A direct blow may break the AT, but about 60% of injuries occur in amateur athletes pushing off with a straight leg.[1] This group usually tears close to the tendo-muscular junction, with better healing prospects than those of older, debilitated subjects, whose tear is usually in the distal, avascular part of the tendon.[2] The patient was gratified to learn he had joined the former group.

At the time of injury the left ankle was carrying a static load of about 125 kg, increased by the forward thrust and perhaps by the boat motion. Clearly a loaded diver making a stride entry is a candidate for injury. A broad platform for a diver's take off would avoid over-extension of the tendon and seems a reasonable recommendation. When heavily laden, entry from a sitting position may be less stylish, but removes the risk of ankle injury.

Diagnosis

Symptoms may be misleadingly minor. A false positive diagnosis of AT rupture may be reached with pain and weakness from a torn Gastrocnemius or Plantaris, or occasionally with acute inflammation of an accessory Soleus muscle lying between AT and tibia.[3] Sometimes a patient may report an audible pop as the tendon breaks, but often, as in this case, there is neither sound nor much pain. There is always weakness of ankle extension, though full action remains thanks to intact Plantaris and long toe flexors. This residual action may encourage an element of denial. Sometimes there is a palpable gap in the tendon, but this may be masked by local swelling.[4] These factors explain the reported 20-30% of missed diagnoses.[5]

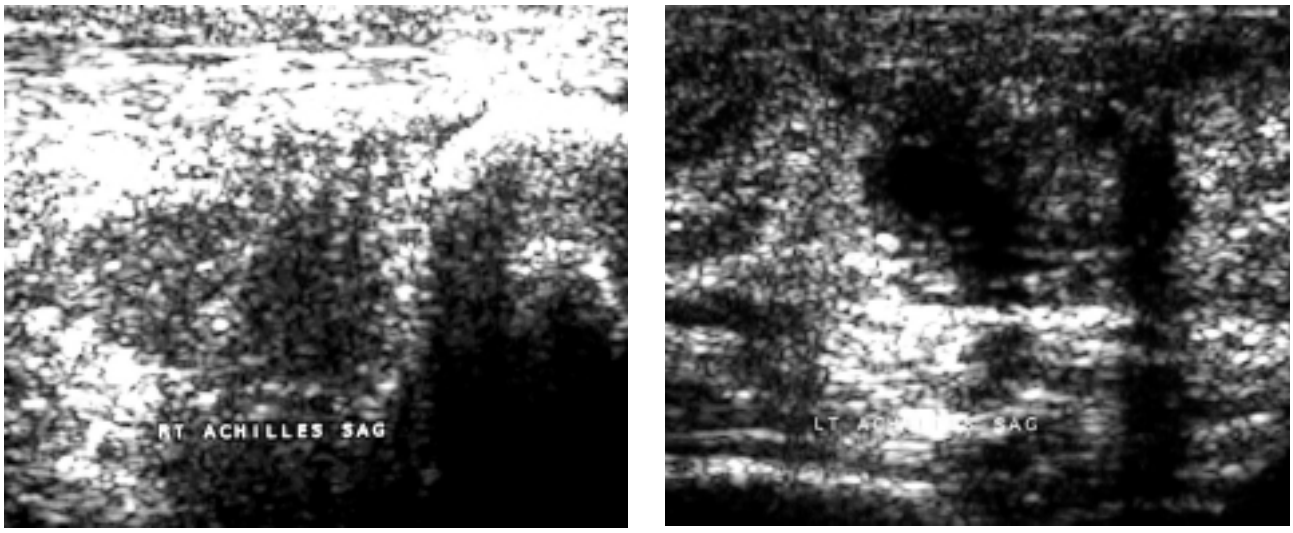

Figure 1. Ultrasound scans of the right intact Achilles tendon and of the ruptured left tendon. Scans and reproductions by Dr R Jones.

Early diagnosis is very desirable, since four weeks' delay in treatment may leave a 20% loss in muscle power.[5,6] Imaging by a skilled ultrasonographer can show the tendon gap, and on ankle extension the small tendons may be seen to slide past the static AT. Magnetic Resonance Imaging is sometimes available for those who can afford it, though luckily there is a reliable and less expensive test that has been available on every dive boat since it was first described thirty-six years ago.[7] This, the Thompson test, requires the patient to lie prone with the knee up at right-angles. When the calf is squeezed firmly an intact TA extends the ankle against gravity. There is no movement from a broken tendon.

Treatment

Tendon healing is by migration of tendoblasts from damaged fibres and laying down of new collagen, but also by in-growth of vessels and fibroblasts from surrounding connective tissue.[8] Treatment is aimed at minimising the latter, gap-filling process by early apposition of the broken ends. Inactive patients may be offered conservative treatment, with the foot immobilised in equinus, though at the cost of a weakened union. Open repair, using heavy, non-absorbable sutures, is preferred, either with or without fascial graft reinforcement. After removal of the cast a brace with dorsiflexion stop will curb over-enthusiastic mobilisation.[9]

Conclusion

Amateur athletes are a recognised at-risk group for TA rupture. No doubt the diving fraternity will be pleased to be counted among them, though other pre-disposing factors, obesity and sub-optimal fitness are, regrettably, always with us. Gout is said to make the injury more likely, as is previous, well-meant, injection of steroid.[10] In these enlightened days of diving for the disabled perhaps we should be prepared also for other listed factors, rheumatoid arthritis, subacute lupus erythematosus, diabetes mellitus, renal failure and hyper-parathyroidism.

The rarity of TA rupture as a dive injury is hard to explain. Neither the SPUMS Journal Index (covering the years 1972-1998) nor Dr Douglas Walker, Collator of Project Stickybeak, has a reported case. This rarity, plus the undeserved nature of many ruptured TA injuries are clearly factors in the common diagnostic delay. A denial factor and a low index of suspicion play a part as well. The take-home message must be that rapid referral will minimise morbidity. Finally, as illustrated by the case described, our more obsessive patients have to be impressed with the need for patience to achieve firm healing before they return to full activity.

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Acknowledgments

The author thanks Dr Nick Cooling, Mr John Mills and Dr R Jones of Hobart, for diagnosis and treatment of the case described, and Ms Anne Batt, Medical Librarian, University of Western Australia, for research assistance.

Dr J C Marwood MRCS LRCP is a General Practitioner. His address is 8 Sloane Street, Hobart, Tasmania 7004, Australia. Telephone +61-(0)3-6223-7887 or +61-018 047 806. Fax +61-(0)3-6228-9530.

THE WORLD AS IT IS

WHY DID DAN SUSPEND COVER FOR VANUATU ?

John Lippmann

Key Words

Accident, decompression illness, insurance, rescue, transport.

Over the past two years, DAN has been involved in the evacuation of around 15 divers from Vanuatu. We are also aware of a substantial number of divers who were not evacuated but who required treatment for decompression illness after returning home from Vanuatu.

Many of these divers had been diving on the *President Coolidge*, off Santo, to depths from around 20 m to approaching 70 m. DAN has expressed concern for some time about the amount of deep diving conducted on the *Coolidge*. In particular we have been concerned about:

1 The depth and number of dives conducted;
2 the minimal experience of many of the divers;
3 the incidence and severity of decompression illness occurring;
4 the lack of appropriate medical facilities for treating divers in Vanuatu;
5 the general lack of appropriate oxygen equipment and trained personnel;
6 the difficulty and delays sometimes associated with evacuating a person from Vanuatu;
7 the cost of evacuating a person from Vanuatu (around $40,000).

Many divers believe that DAN is little more than an insurance company. **DAN is not an insurance company**. DAN buys insurance for its members from insurance companies. DAN is a non-profit, membership-based organisation dedicated to improving dive safety though a variety of activities that include:

1 Regular dive safety journals; educational programs and seminars;
2 funding diving emergency hotlines (e.g. the DES in Australia);
3 providing evacuation cover;
4 providing very economical dive injury insurance;
5 providing economical and effective oxygen equipment;
6 the provision of advisory services for diving health and safety;
7 providing support for the establishment of recompression chambers;
8 donation of safety equipment;
9 collection, analysis and reporting of dive accident data.

Over the past 12 months or so, DAN SEAP has expressed its concern to several dive operators in Vanuatu, pointing out that some insurers were considering placing restrictions on cover for certain dive destinations known to produce DCI. We warned that it was possible that some insurers might cease to offer cover for diving accidents in certain places. Despite our expressions of concern, we observed no reduction in the number of cases.

DAN believes that the best solution would be to have a suitable recompression facility established in Vanuatu. To this end, we approached some organisations and requested that they consider establishing such a facility. DAN offered to make a substantial initial contribution to the chamber and has offered to provide some funds to cover the on-going training and supervision requirements. One of the organisations we approached has spent considerable effort investigating establishing a facility in Vanuatu. The essential condition is that chamber is financially supported by the dive operators, who would have to put a small levy on the cost of each dive to cover the ongoing maintenance and operational costs of the chamber. There was some initial reluctance of some dive operators to support the levy concept and the potential chamber installer has notified us that he is now unwilling to commit himself to installing a chamber in Vanuatu.

The cost of evacuating divers from Vanuatu has been so high that our insurers threatened to cease offering us cover for evacuations from anywhere else, which could affect the viability of DAN S.E. Asia-Pacific. Clearly, we cannot risk our other vital services. As a result, we have been forced to suspend cover for evacuation from Vanuatu. Although we will cover divers who were DAN SEAP members at the time we restricted cover (July 1998), no new or renewing members will be covered for Vanuatu. This situation will be reviewed if and when an appropriate recompression facility is established in Vanuatu.

DAN SEAP has always advised its members to take out appropriate travel insurance when travelling overseas to cover a variety of situations, such as medical treatment, loss of luggage and so on. Many people feel far more comfortable having DAN involved in the evacuation, rather than an organisation that may not be as experienced in managing dive accidents. If a DAN member has travel insurance that will cover a diving accident we will be happy to work with the insurer, if we are contacted by the member, to advise on and facilitate an evacuation if required. However, many travel insurance policies impose a depth limit of 40 m and do not cover diving deeper.

DAN SEAP works hard to improve dive safety services throughout the Asia-Pacific region. For some years we have been involved in training the diving industry in accident management and have facilitated the availability of first aid equipment and facilities throughout the Region. We have established National offices in the Philippines (Makati Medical Centre), New Zealand (NZ Naval Hospital) and soon in Malaysia (Naval Hospital) to improve the availability of treatment to both residents and visitors to those countries. We have provided support for the establishment of recompression facilities in Phuket, Thailand and Papua New Guinea. The latest extensions of DAN SEAP's work are the Nationally Accredited DAN SEAP general and workplace first aid courses that are now available in Australia.

DAN works hard to support divers, and without the support of divers, we cannot continue to function. DAN SEAP works on a very small budget and its few staff work long and hard to assist both members and non-members. The staff get rather frustrated when people see us a "just another insurance company". We are not and will never be such a company. DAN SEAP receives no government funding and receives minimal donations from the diving industry. Everything we do, we fund through membership revenue and certification fees. We need the support of members to exist and to continue to grow. If you believe that DAN is a worthwhile organisation you should encourage other divers to join and find ways to help us. The ball is in your court!

John Lippmann is Executive Director of DAN S.E.Asia-Pacific. He is co-author, with Stan Bugg, of THE DIVER EMERGENCY HANDBOOK which first appeared in 1984 and has since been adopted by DAN USA and the British Sub-Aqua Club. It has also been translated into Japanese, German and Italian. He is also author of DEEPER INTO DIVING, THE ESSENTIALS OF DEEPER DIVING and OXYGEN FIRST AID FOR DIVERS. For his contributions to diving safety he was elected a member of SPUMS in 1996.

The address of DAN S.E.Asia-Pacific is 49A Karnak Rd (PO Box 384) Ashburton, Victoria 3147, Australia. Phone +61-3-9886-9166. Fax +61-3-9886 9155. E-mail danseap@danseap.com.au .
Web page www.danseap.com.au .

SPUMS NOTICES

ERRATUM

***SPUMS J* 1998; 28 (1)**.

In the paper by Carl Edmonds *Drowning syndromes: the mechanism* (*SPUMS J* 1998; 28 (1):2-9) reference 1 (Donald WK. Drowning. *Brit Med J* 1995; (2): 155-160) was misdated. The paper was actually published in 1955 and the date, volume and pages should read 1955; 2: 155-160.

Key Words
Notice.

PROFESSOR JOHN WILLIAMSON AM

The Executive Committee of SPUMS congratulates Dr John Williamson, Director of the Hyperbaric Medicine Unit at the Royal Adelaide Hospital, on his appointment as a Member of the Order of Australia (AM) in the Queen's Birthday Honours list.

The appointment was for his contributions to the fields of Marine Envenomation, Surf Lifesaving, St John's Ambulance and Hyperbaric Medicine. Dr Williamson has also found time to be a Committee Member of SPUMS (1984-1987, 1990-1993 and 1994-19960).

MINUTES OF THE ANNUAL GENERAL MEETING OF SPUMS

held at the Palau Pacific Resort on 15/5/98 at 1610

Apologies

Drs A Slark and J McKee

Present

All members attending the Annual Scientific Meeting

1 Minutes of the previous Meeting:

Minutes of the previous meeting have been published (SPUMS J 1998; 28 (1):16).

Motion that the minutes be taken as read and are an accurate record. Proposed Dr D Gorman, seconded Dr V Haller. Carried.

2 Matters arising from the minutes:

None.

3 Annual reports

President's Report (Printed below)

4 Treasurer's Report

The Auditor's report and Statement of Receipts and Payments are printed on page 199.

Motion that the financial statements be accepted. Proposed Dr I Seppelt, seconded Dr D Davies. Carried.

7 Annual subscriptions

Motion that the subscriptions remain the same (Full and corporate membership $100 and associate membership $ 50). Proposed Dr D Gorman, seconded Dr J Knight. Carried.

6 Election of office bearers

Nominations had been received as follows.

President	Dr Guy Williams
Secretary	Dr Cathy Meehan
Treasurer	Dr Timujin Wong
Editor	Dr John Knight
Public Officer	Dr Guy Williams
Education Officer	Dr David Davies
Committee Members	Dr Chris Acott
	Dr Vanessa Haller
	Dr Robyn Walker

There being no other nominations they were declared elected.

7 Appointment of the Auditor

Motion that Dr Douglas Porter, FCA, of Newport Beach, New South Wales be reappointed as auditor, proposed Dr G Williams seconded Dr J Knight. Carried

8 Business of which notice has been given

8.1 That Dr Anthony Slark be elected a Life Member of the Society. Proposed Dr D Gorman seconded Dr G Williams. Carried

8.2 That Rule 22(b) be altered to increase the period of election to the Committee to three years. The actual wording of the amendment is as follows. That Rule 22 (b), which reads "Each officer of the Association shall hold office until the annual general meeting next after the date of that person's election but is eligible for re-election.", shall be changed by replacing the word *next* by the words *three years*.
The rule would then read. "*Each officer of the Association shall hold office until the annual general meeting three years after the date of that person's election but is eligible for re-election.*"
Proposed Dr G Williams, seconded Dr R Walker.
This motion for spoken for by Dr G Williams, and against by Dr J Knight.
The motion was passed with a show of hands, by a small margin. The motion will be printed in the journal so that members may request a postal ballot.

Closed 1640

Key Words

Meeting.

PRESIDENTS REPORT

Thank you for attending SPUMS' 26th Annual Scientific Meeting, and our third visit to Palau. I would like to thank David Elliott and John Bevan for their participation in this year's meeting, and especially note David Elliott's elevation to life membership at last year's meeting in New Zealand. Chris Acott deserves a special mention for his work in convening this year's meeting, and for his stamina in also being convener for the 1999 meeting in Layang Layang, Malaysia. I hope many of you can attend next year's meeting which will be held in the first week of May. Our guest speakers will be Alf Brubakk and Richard Moon, the topic will be Bubbling, with a workshop on the Clinical Aspects of the Treatment of Decompression Illness.

This year sees the birth of SPUMS on the Net, members and non-members will be able to obtain information on SPUMS and Diving Medicine, membership information, details of our meetings and the SPUMS Journal, on line. I hope this will increase our exposure to the world and result in a boost to our numbers outside Australia and New Zealand.

Guy Williams
President SPUMS

AUDIT REPORT
TO THE MEMBERS OF THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

I have conducted various tests and checks as I believe are necessary considering the size and nature of the Society and having so examined the books and records of the South Pacific Underwater Medicine Society for the year ended 31 December 1997 report that the accompanying Statement of Receipts and Payments have been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the period then ended.

17/9/97 David S Porter
361 Barrenjoey Road, Newport Beach, New South Wales 2106. Chartered Accountant

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

STATEMENT OF RECEIPTS AND PAYMENTS FOR THE YEAR ENDED 31 DECEMBER 1997

			1997		1996
Opening Balance					
ANZ Bank	1995 ASM Fiji	-		11,090	
	Access Accounts	32,041		(3,722)	
	ANZ V2 Plus	68,045		92,320	
			100,086		99,688
Receipts					
Subscriptions and registrations		92,518		161,430	
Interest		4,388		5,707	
Advertising and Journal sales		402		1,201	
Sundry income		210		1,455	
			97,518		169,793
			197,604		**269,481**
Payments					
ASM costs		6,161		20,393	
Travel		-		22,779	
Secretarial		4,754		10,865	
Stationery and Printing		1,198		5,183	
Journal		27,554		36,792	
Postage and facsimile		4,459		6,074	
Conferences and telephone		6,952		9,324	
Computer equipment		8,281		36,300	
Miscellaneous and subscriptions		2,223		952	
Bank Charges					
2,151		2,604			
Audit		1,100		1,080	
Insurance		-		799	
Editor’s honorarium		15,300		16,250	
Oxydive kit		2,561		-	
			82,694		169,395
Closing balances					
ANZ Bank	Access Cheque Account (new)	1,740			
	Access accounts	33,170		32,041	
	ANZ V2 Plus	80,000		68,045	
			114,910		100,086
			197,604		**269,481**

These are the accounts referred to in the report of D S Porter, Chartered Accountant, Newport Beach, NSW 2106, dated 17/9/97

MINUTES OF THE
SPUMS EXECUTIVE COMMITTEE MEETING
held at the Palau Pacific Resort on 11/5/98 and 15/5/98

Present

Drs G Williams (President), D Gorman (Immediate Past President), C Meehan (Secretary), T Wong (Treasurer), J Knight (Editor), D Davies (Education Officer), C Acott, V Haller, R Walker (Committee members), H Straunstrup (European Representative) and M Bennett (ANZHMG Representative), on 15 May only.

Apologies

Dr M Kluger (NZ Representative)

1 Minutes of the previous meeting

Minutes of the previous meeting on 1 November 1997 read and accepted as a true record after minor adjustments. Proposed Dr J Knight, seconded Dr R Walker. Carried.

2 Matters arising from the minutes

2.1 North American Chapter. Mr Jeffrey Bertsch will be the chapter representative. The financial affairs of the chapter prior to now are considered closed.

2.2 Indemnity Policy update. Dr Williams will be following this up.

2.3 Job description update of the Editor, Treasurer, and Convener. An updated job description of the Editor and the Treasurer has been received. The job description of the Convener is still pending.

2.4 Oxygen equipment update. It was not necessary to take oxygen equipment to Palau as there was ample equipment available.

2.5 Upgrade of the audio equipment. All the remote microphones have been upgraded. Dr Acott provided information on a SVGA projector. The committee will review the specifications and further discussion will be held.

2.6 SPUMS on the Internet. There have been small technical problems in setting up the domain site. However, it is expected that this will be resolved shortly. The domain name is http// www.SPUMS.org.au It is suggested that this site is viewed and any errors are corrected before it is posted. Any additional information or links that should be on this site should be sent to the Secretary.

2.7 New application form will be printed after the ASM. Committee members are invited to comment on the layout of the new forms. The Treasurer would like the application form and renewal notice to be on one form. The benefits of this and enclosing the renewal notice with the December journal should be discussed further.

2.8 Secretariat. It was decided that the Secretary and Treasurer should continue to use SPUMS paid typists.

2.9 ANZHMG. The chairperson of the ANZHMG will be invited to attend all the SPUMS Executive Committee meetings, including the face to face meeting held towards the end of every year. The chairperson of the ANZHMG is to be the recognised spokesperson for all matters associated with HBO therapy. It is thought that a motion to change the constitution to formalise this arrangement will be put forward at the 1999 AGM. The wording of this motion is yet to be decided.

3 Annual Scientific Meetings

3.1 1998 Palau update. There will be a meeting between the Convener, the Treasurer and the Allways Dive Expedition representative. All the outstanding expenses will be discussed. There will be discussion as to the appropriate allocation of the FOCs.

3.2 1999 Layang Layang update. The workshop at this meeting will be the Treatment of Decompression Illness. There were no other outstanding matters to discuss.

3.3 2000 proposed Fiji. It has been suggested that this conference be a family orientated conference as was the case in 1995. The proposed Conveners for this meeting are Drs G Williams and V Haller.

3.4 2001 proposed PNG Kavieng. It was proposed that Dr C Meehan be one of the convenors.

4 Treasurer's Report

The Treasurer's report was presented and the Society is in good financial standing. In view of this there is to be no change in the subscription fees this year.

5 Correspondence

5.1 Letter from Bob Ramsay re HBOT future. This matter will be passed onto Dr M Bennett (chairperson of ANZHMG).

5.2 Letter from John Knight re registration fee for accompanying persons. This was discussed.

6 Other Business

6.1 It was discussed and agreed that, in future, the conference conveners should have their airfares and accommodation covered by some of the allocated FOCs. They would no pay the registration fee. In the past they have paid all their own expenses.

It was agreed that the President, Secretary, and Treasurer, and other Committee Members should not be financially assisted to attend the Annual Scientific Meeting .

6.2 Proposal with regards Toshiba notebooks when warranty runs out early 1999. No decision was made on this.

6.3 Revision of AS4005.1 and revision of the SPUMS Diving Medical. As AS4005.1 was being revised it was considered necessary to revise the SPUMS Diving Medical on which the AS4005.1 is based. Proposals for changes were given, and these have

been passed to the Australian Standards committee by Dr C Meehan, the current SPUMS representative on that committee.

6.4 Review of the Editor's honorarium. There were no changes made to the honorarium for the coming financial year.

6.5 A membership drive was discussed.

6.6 Dr Henrik Straunstrup, the European Representative, discussed his role and the possibility of having closer links.

Key Words

Meetings.

CONSTITUTIONAL AMENDMENT

At the Annual General Meeting held in Palau on 15/5/98 the following amendment to the constitution was passed.

That Rule 22 (b), which reads "Each officer of the Association shall hold office until the annual general meeting next after the date of that person's election but is eligible for re-election.', shall be changed by replacing the word *next* by the words *three years*.

(Continued on page 202)

(Continued from page 201)

The new wording would be: *Each officer of the Association shall hold office until the annual general meeting three years after the date of that person's election but is eligible for re-election.*

As no member has notified the Secretary of SPUMS that he or she objected to the above amendments it is assumed that the Membership has voted in favour of the above amendments which now come into force

Cathy Meehan
Secretary of SPUMS

Key Words
Constitutional amendment, notice.

1999 SPUMS ANNUAL SCIENTIFIC MEETING

will be held on the island of
Layang Layang,
Malaysia

Friday April 30th to Sunday May 9th 1999

The Guest Speakers will be Dr Richard Moon (USA), who was a guest speaker at the 1997 ASM at Waitangi in New Zealand and Dr Alf Brubakk (Norway), who attended the 1998 ASM in Palau

The Convener of the Annual Scientific Meeting is Dr Chris Acott. The provisional title of the theme of the meeting is *Gas bubble injury and its treatment.*

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

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

The Official Travel Agent for the meeting is:
Allways Dive Expeditions
168 High Street, Ashburton, Victoria, Australia 3147
Telephone + 61-(0)3-9885-8863.
Toll Free with Australia 1-800-338-239
Fax +61+(0)3-9885-1164.
Email: allways@netlink.com.au

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

DIPLOMA OF DIVING AND HYPERBARIC MEDICINE.

Requirements for candidates

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

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

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

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

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

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

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

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

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

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

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

Key Words
Qualification

LETTERS TO THE EDITOR

THE LADY IN THE PRESIDENT'S SMOKING ROOM

201 Wickham Terrace
Brisbane
Queensland 4000
6/10/98

Dear Editor

It is every diver's ambition to dive on the *President Coolidge* at Santo, the world's largest accessible sunken liner.

The Lady (her portrait is below) is the highlight of diving on the *President Coolidge* and lies at a depth of 45 m (150 ft). When I first saw the art work through the gloom of the torchlight two years ago one could not be blamed for thinking one was slightly "narked". The unusual relief consists of a standing woman in Elizabethan garb next to a unicorn (now minus its horn). The fireplace ornament was first found by Allan Power in 1981.[1] Apparently it had been protected by a timber covering when the liner was converted to a troopship. In the years since the ship struck a US minefield, on October 26th 1942, the timber had rotted away.

During a second dive to *The Lady* it was examined in more detail and Bettina and I questioned the significance of a combination of an Elizabethan woman standing next to a unicorn. To our knowledge none of the written material about the wreck discuss this enigma.

According to myth a unicorn is seen only by virgins or women of gentle nature. The *President Coolidge* was launched in Virginia ,which was named by Sir Francis Drake in honour of the Virgin Queen, Elizabeth 1.

We propose that the female figure in fact represents Queen Elizabeth 1. This would explain her gown, the full ruff as worn by an unmarried woman and also her reddish hair, albeit slightly faded.

What we believe seals the argument, is the presence of what almost certainly represents two Tudor roses, one above *The Lady's* left arm and another below the unicorn's left front hoof. The Tudor rose was, of course, the family emblem of Elizabeth 1.

In any event *The Lady* warrants at least one visit should one have the opportunity of diving to the first class passengers' smoking room on the *President Coolidge*. For the careful and experienced diver it is a safe and rewarding dive.

William Douglas

Acknowledgments

Photograph courtesy of Steve Hills, Prodive, Milton, Brisbane, Queensland.

Reference

1 Stone P. *The Lady and the President*. Melbourne: Ocean Enterprises, 1997

Key Words

Environment, general interest, history.

DAN SEAP INSURANCE COVER AND VANUATU

201 Wickham Terrace
Brisbane
Queensland 4000
15/10/98

Dear Editor

The recent decision by DAN S.E.Asia-Pacific to discontinue evacuation cover for Vanuatu does not surprise me. I have treated three divers with decompression sickness and one with near drowning at the Santo Hospital. The conditions in Santo Hospital are atrocious and I cannot imagine anybody who has the experience to run a decompression chamber safely, staying in that town to run a chamber.

The quality of medical care in Vila is a good deal better and so, if a chamber is established, Vila would be the most suitable site. Obviously this involves transfer of divers from Santo to Vila. However, this is a much easier process than bringing a plane in from Melbourne, or anywhere else and transporting them back to Australia.

I found dealing with the Insurance Companies (not DAN) to be a most frustrating affair and it took approximately 17 hours for the retrieval team to collect the injured divers.[1]

It may be appropriate for you to approach DAN SEAP to comment on this matter as hundreds of divers will continue to visit the *President Coolidge* every year and further diving related injuries are certain to occur.

William Douglas

Reference

1 Douglas W. Travel insurance for divers. *SPUMS J* 1997; 27 (1) March: 33-34

Key Words

Accident, decompression illness, insurance, letter, rescue, transport.

*Readers are referred to the **WORLD AS IT IS** for further comments about Vanuatu.*

BOOK REVIEWS

BOVE AND DAVIS' DIVING MEDICINE 3RD EDITION.
Editor A A Bove.
Philadelphia: W B Saunders Company
Review copy provided by Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price from Best Publishing Company $US 77.00. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail divebooks@bestpub.com .

There are three well known books in English on Diving Medicine that all those seriously interested in Diving Medicine should have on their bookshelves. **This is one of them** and the other two are *The Physiology and Medicine of Diving*, edited by Bennett and Elliott, from England and *Diving and Subaquatic Medicine* by Edmonds, Lowry and Pennefather from Australia. The latter, now in its 3rd edition (1992), is a very clinically orientated volume of 565 pages (reviewed SPUMS J 1992; 22 (2): 90). Bennett and Elliott, now in its 4th edition (1993), is a well-edited, multi-author production of 613 pages which appears to be largely aimed at those who care for, and supervise, naval and occupational divers (reviewed SPUMS J 1993; 23 (3):157-158).

Bove and Davis is another multi-author volume, this time of 418 pages, which tries to provide for both audiences. On the whole this approach has succeeded. There are some places where the editor has not been sufficiently busy with the red ink and allowed considerable repetition, but then it is well known that only about half of what one is told is usually taken in. Repeating things should increase the chances of the information being absorbed.

The list of 26 authors contains the names of 8 who have been guest speakers at a SPUMS Annual Scientific Meeting, one three times and three twice. Bennett and Elliott, with 22 authors have 6 guest speakers. Nine of the authors have contributed to both books. This makes it clear that this is a mainstream book with the chapters written by experts. The vast majority of the book is widely accepted and clearly presented. One of the pleasures of reading the book for this review was the constant use of the term "alternative air supply" rather than the PADI usage of "alternate air supply", which suggests swapping between the primary regulator and the octopus, breath by breath, throughout the dive.

What is surprising is that a scientific book on diving medicine published in 1997 should contain a chapter on Diving Physics where the calculations are in "imperial" measurements, feet of sea water and cubic feet. Certainly they are repeated in SI measurements but all the rest of world has long ago converted to metric for scientific work. Perhaps that is a bit unfair, if the book is solely designed for the US market, where Clive Cussler's dream of a metric America, for Dirk Pitt to carry on with his adventures, seems destined for non-fulfilment. But the book should be read around the world for the different slants it put on various aspects of diving medicine. On the other hand Bill Hamilton's chapter on Mixed Gases is entirely metric, and as a result the calculations were much easier to follow.

The chapter on the Mechanisms and Risks of Decompression is surprisingly blinkered. Only the Haldanian theories are discussed in any depth and no mention is made in the text, or references, to the work of Brian Hills and the thermodynamic theory of decompression. Applying this theory has shown that deep stops can increase diver safety and speed decompression. Hills' book Decompression Sickness Volume 1 (there never was Volume 2) was published in 1977! Another omission is any mention of the fact that washout of gas after a dive is

not the mirror image of uptake, although most Haldanian decompression calculations assume that it is. Luckily the following chapter Pathophysiology of Decompression Sickness gives a wider view of the processes and problems of decompressing from a raised environmental pressure.

The excellent chapter on Hypothermia, covering the full spectrum from mild to serious, is apparently aimed at hospital doctors as, beyond simple insulation, there is no treatment (rewarming) recommended until the "the core organs" have been warmed by peritoneal lavage. There is a good review of the problems to be expected when warming a person in a bath and the modern view that the arms can be immersed without increasing risk is given mention. With mild hypothermia being very common among divers in cold water, while moderate hypothermia is uncommon and severe rare, after sensible exposures or when using dry suits, one would expect some advice aimed at the rapid treatment of rapid-onset, mild and moderate hypothermia.

The chapter on Pulmonary Disorders has a good discussion on asthma and diving but, although methacholine and histamine challenges are mentioned hypertonic saline challenge is not. The SPUMS 1995 ASM concluded that hypertonic saline has a place in the assessment of asthmatics for diving.

The chapter on the Medical Evaluation for Sport Diving is excellent and largely as the late Jefferson Davis, who in 1979 was the second SPUMS guest speaker, left it in 1989 with some changes to reflect present day views provided by Fred Bove. It is followed by David Elliott dealing with the same topic for commercial diving with a brilliant precis of Edinburgh conference in 1994 on *Medical Assessment of Fitness to Dive* (reviewed SPUMS J 1995; 25 (2): 72-73).

The final chapter is a discussion of US Navy (USN) Diving Techniques and Equipment. It presents a fascinating collection of information, only slightly obscured by using scuba to describe all self-contained underwater breathing apparatus. This might have been acceptable in the 50s and 60s but in the 1990s scuba has come to mean open circuit air apparatus. Luckily the author more often uses UBA (underwater breathing apparatus) when discussing rebreathers. The USN adopts a less safety conscious attitude to oxygen sets than the Australian or British navies, who use full face masks with all rebreathers. The USN only uses full face masks for their Mark 16 UBA, the $US 34,000 mixed gas rebreather with multiple electronic sensors to control oxygen levels.

John Knight

Key Words

Book review, decompression illness, diving medicine, equipment, physiology, treatment.

SPUMS ANNUAL SCIENTIFIC MEETING 1998

DECOMPRESSION THEORY IN THIRTY MINUTES

David Elliott

Key Words

Decompression illness, history, occupational diving, physiology, tables.

Introduction

The recognition of pressure-related illnesses in divers and compressed air workers and the first ideas on the control of those hazards evolved almost blindly and with little scientific direction during the 19th century. A long time passed after the publication of Paul Bert's work in 1878 before there was any real recognition of its message by the worldwide scientific community.[1] The pioneering applied science of John Scott Haldane began some 25 years later.[2] Haldane focussed upon the applied physiology of diving while, at the same time, Sir Leonard Hill was making the first quantitative analyses of nitrogen in blood and urine at pressure.[3] Hill favoured the ΔP concept, a constant pressure difference for the linear decompression of caisson workers. Each physiologist made contributions to both diving and compressed air work but, in spite of arising from a common stem, the subsequent development of safe decompression procedures for the shallow but prolonged dry exposures of compressed air workers has followed a different path from that for divers. The lessons to be learned by divers from caisson work today are few and, if anything, the transfer is now in the other direction. It is therefore the purpose of this brief review to focus on aspects of the development of decompression theory and confine that to only diving. And somehow all this has to be done in a time slot that would make a single evening for the condensed performance of all Shakespeare's plays seem generous.

To maximise the benefit, if any, from such a speedy approach it might be helpful to sketch out the route now be followed and identify some of the features to be spotlighted. Those who then wish to read more deeply in this subject with either a detailed research review,[4-7] or simply an advanced instructional text,[8] can use this brief overview as a guide to some practical difficulties that often seem overshadowed by computational wizardry.

Impossible variables

Decompression theory is readily amenable to mathematical modelling but the reality of trying to apply this theory is the basic problem which has bedevilled all research in this field, that of inter-individual and intra-individual variation. An example of the first is that "4% of the workforce get 50% of the bends"[9] and of the second is the common observation of a diver getting "hit" on a much safer profile than usually dived. What Haldane and his successors have done so effectively over the last 90 years for the safety of the diving population as a whole does not necessarily hold true for you on your next dive.

The presence of biological variation was acknowledged in some of the early studies but, at the beginning of this century, the morbidity was so gross that such subtle considerations were not necessary in order to make considerable progress. In later years, decompression scientists have reviewed some of the relevant factors but have not been able to integrate them into their mathematical models. Not surprising when one has to consider not only the effects of exercise at depth and during decompression, of hot water at depth and cold during decompression upon gas dynamics, but also all the individual factors, from age to hydration that may be related to susceptibility. Indeed, the pessimist can reach the conclusion that one mathematical model, however complex, for all divers is an impossible target, just on the basis of no more than the evidence of one case of a successful response to therapeutic recompression of a knee "bend" which followed some 30 min after a 5-minute bottom time dive to 100 feet (30 m). That bend, and there are many like it, is outside the predictive models used for regular tables and has to be handled in some other way. Probabilistic tables can cope with this extreme phenomenon but, of course, may be too long to be practical and of no consolation to the one diver who does get hit. Figure 1 (on page 211) shows the wide variability of no-stop curves (mild bends end point) in goats.[5]

Another underlying problem can be summarised by the title chosen by Nashimoto for a UHMS Workshop:[10] *"What is Bends?"*. There are no internationally accepted criteria to define the boundary between a dive that is clean and one that is not. What indeed was meant by different authors by the term "bend" when one reads reports on the bends incidence of different decompression procedures at different times and in different locations? Until Nashimoto's question *"What is bends?" can* be answered precisely, there is also no answer to the one question which is the foundation of today's quest for decompression safety, *"What is a safe decompression?"*

The main end-points that have been used in decompression studies in man over the years have included:

1 **reported symptoms, and physical signs** if detected, but these are largely dependent on the individual diver's reporting threshold. Severity ranges from vague "niggles" in the joints to a life-threatening illness, so where is the end-point to be defined?

2 **bubble counts in man**. Used successfully for the comparative testing of the Canadian tables[11,12] but not always reliable for diagnosing decompression illness.[13]

3 **recompressions**. Where a chamber is readily available, this is a definite event but, in addition to the reporting threshold of the diver, recompression is dependent also on the interpretation of the diver's symptoms by the chamber operator and/or diving doctor,

4 **long term outcome**:[14] ranging from neurological residua to bone necrosis.

Also to be considered in the development of decompression tables are the methods by which they were evaluated, with what subjects, under what circumstances and, again, how the end-point was assessed. Early naval tables were tested by sample profiles being dived under careful supervision by a relatively small team of fit young divers, well acclimatised to this type of diving. The target at RNPL in the development of deep helium bounce and repetitive air dives in the 1960s was to achieve "10 clear dives", defined by no recompressions. It was later calculated by Homer and Weathersby that as many as 40 dives might not indicate anything more precise than a bends risk for the dive of somewhere between 17 and <1%.[15]

Even when using a consistent end-point, the "bends percentage" is potentially misleading. If 10 men each perform 100 dives on a specified schedule and between them, in those 1,000 dives, there are 10 episodes of bends, then there can be alternative ways of presenting this 1% exposure rate.[9] By most table and probability assumptions, this risk is considered to be evenly distributed among the diving population. This means that at some time each diver would have had one bend and so this would be, for the 10 men involved, a 100% bends rate. Given the association between a history of recompression and the later development of osteonecrosis, this could be a more meaningful figure. If, at the extreme of biological variation, one susceptible man had all 10 bends then the best figure for this trial is that 10% of the divers were affected, but it is still 1% of exposures, the most commonly used index.

After the acceptance of a "tested" naval table into use, the subsequent reported bends incidence at sea may be different and this may be a reflection of procedural differences between the meticulous testing of the printed tables and the way in which operational dives are actually performed. Indeed, the tables may be safer "as used" because of the introduction of additional safety factors on site when estimating depth and duration.

Decompression table testing

In place of the linear decompressions recommended by Bert and von Schrotter,[1,16] a staged decompression was introduced by Haldane[2,17] based on 5 hypothetical compartments in the body (misleadingly called "tissues") with half times for gas uptake or elimination of 5, 10, 20, 40 and 75 minutes. The latter tissue was chosen because a 75-min tissue becomes 95% saturated in 5 hours and, as advised by E S Moir, compressed air workers did not appear to get an increased number of bends once they exceeded 5 hours at pressure. Haldane recommended an initial decompression, provided that this was from less than 6 atmospheres absolute (i.e., a depth of 50 m), to half the absolute pressure. This was then followed by the appropriate series of predetermined stops. In the same report it was recognised that an inadequate flow of air causes a build-up of carbon dioxide in the diver's helmet and that it was necessary to increase the minute volume in proportion to the increased pressure of depth. The Haldane tables were adopted by the Royal Navy in 1908 and subsequently adapted by the US Navy who added a 120-min half time compartment giving 98.5% saturation in 12 hours. Stillson also reduced the Haldane 2:1 ratio to 1.58:1 and included the option of oxygen decompression in these USN "Construction and Repair" Tables.[18]

In the UK , Damant and Davis also reduced the ratios and, for dives between 120 and 330 feet (36 and 100 m), introduced oxygen stoppages.[19] In the USA, Hawkins, Shilling and Hansen analysed several thousand dives and concluded that the faster compartments could tolerate higher ratios.[20] Their subjects made daily dives, 5 days a week, with 8 subjects at each depth and time of exposure. The stated end-point in each of these runs was the "production of caisson disease of severe enough nature to necessitate terminating the series".

Yarborough revised the Construction and Repair table,[21] eliminating the 5 and 10 minute tissues, and these resulted in a bends incidence of 1.1% but, as discussed, this percentage is not necessarily comparable with rates from other tables or other locations.

During the Second world War, there was much basic research on bubble nucleation and growth and on the patho-physiology of decompression in relation to high altitude and diving, excellent work that still repays reading.[22] After the war the Yarborough tables were reassessed and a very much higher bends incidence was found on deep dives. It was concluded that, to control decompression on the deeper stops, the 5 and 10 minute compartments should be reinstated.[23]

The US Navy also developed 'surface decompression' tables,[24,25] seemingly unaware of the experience of this procedure (*"crash surfacing"*) gained by Wotherspoon's divers in the cold waters of the St. Lawrence during salvage operations on *The Empress of Ireland* in 1914 and later by Damant during salvage of *Laurentic*.[26]

Behnke drew attention to a disequilibrium in gas tensions which he termed "the oxygen window".[27,28] US Navy tables were taken further by Workman who used half times of 5, 10, 20, 40, 80 and 120 minutes and revised values for the surfacing ratios.[29] These Workman tables are the basis of most tables which are in use today. To test these dives, 6 "clear" exposures were validated on each of the 88 computed schedules by naval divers exercising in water.[30] Workman subsequently developed the concept of M-values which defined the maximum tissue pressure (M-value) in feet of sea water for each tissue at each stop and for surfacing.[31]

In 1952, Hempleman questioned Haldane's concept of perfusion as the dominant factor for inert gas uptake and proposed a radical new approach:[32,33] a single tissue with diffusion as the rate-limiting factor. Essentially this suggests that the critical excess quantity of dissolved gas = $P\sqrt{t}$, and this provides, where t is less than 100 minutes, no-stop times which are very close to the US Navy's no-stop curve. Using this principle, the consequent RNPL 1968 Air Tables were relatively conservative and, although not popular for that reason among recreational divers, they were used successfully in the UK for deep and arduous working dives.

Hills continued the debate on what he called "*the perfusion diffusion confusion*" by using a thermodynamic model based on outward radial diffusion from a capillary perfusing the length of a hypothetical cylinder of tissue.[5] These and other aspects of his model were expressed in complex formulae, which I consistently failed to master, but his output was a series of ideas and pilot studies, each of which spotlighted contemporary controversial assumptions.[34] Also, in another laboratory while he was at RNPL, the unpredictable dynamics of flow and flow reversal within individual capillaries to be seen in a rabbit ear-chamber were a practical reminder that major individual variations can occur which may be concealed within the averages of large populations.

An exponential-linear uptake and elimination model was then used by the US Navy for the development of "constant PO_2' decompression profiles which have been successfully tested and are the basis for a decompression computer for air diving.[7,35]

Because the naval decompression tables were designed for "square wave dives", they are perceived as penalising the recreational diver by their inflexibility for multi-level dives. The recreational training agency PADI introduced tables with multi-level and repetitive procedures for their recreational divers, who are expected to stay within no-stop limits. Another recreational training agency, BSAC, produced their own tables designed for decompression

diving. However, even in the no-stop range, the majority of recreational divers prefer to use personal diving computers for decompression safety. These provide immediate on-line guidance based on the actual pressure-time profile to that moment. A pioneering analogue computer was based on a mechanical series of compartments[36] but now all personal computers are digital and use a pressure transducer for input. A few computers are based on the established tables, such as those of the US Navy, but most now contain a pre-programmed mathematical decompression model selected by the manufacturer. There are many different computer models of inert gas uptake and elimination is use,[37] some based on modified Haldanean models, some developed by the late Professor Bühlmann (several using a 0.877 bar reference pressure for diving at altitude), some based on the mathematics of bubble growth and resolution, with a few based on yet other concepts, but all have one thing in common, like the tables before them, they cannot cater for the individual purchaser. Each internal computer program must cater for everybody with sufficient "padding" in the computer's calculations to ensure a very low probability of decompression illness in the worldwide buying public.

Although, therefore, most computers will tend be oversafe for the majority of divers, even this may not be perceived by some divers to be safe enough. There may appear to be no obvious way of introducing additional safety factors for a susceptible diver but switching the computer into the altitude mode when diving at sea level merely shortens all the no-stop times, e.g. at 18 m with one computer to 39 minutes (from USN 60 min and DCIEM 50 minutes), and at 30m from 18 minutes in the same computer to 14 minutes (c.f. USN 25 minutes and DCIEM 15 minutes). This is not very popular with one's buddy and, in any case, is merely an arbitrary change to the underlying mathematical model, not a logical one. A more acceptable alternative is to breathe nitrox (oxygen-enriched air; EANx) and then decompress as though on air, but this too says much about our scientific ignorance of determining the safe limits when it comes to planning a safe decompression for the individual.

For an experienced young and fit diver, who wants to avoid being penalised in terms of useful bottom time by what he may perceive as oversafe decompression models designed for the elderly and infirm, purchasing a personal computer with "improved algorithms" will not necessarily be the right answer. The phenomenon of acclimatisation to decompression stress is real but cannot be accounted for in a quantitative manner. Equally real is a fairly common observation that after many dozens of safe but extreme dives, suddenly and for no obvious reason, the same dive again is this time followed by devastating paralysis. Probability theory may be useful but, once the level of risk has been selected, there is little of predictive value about individual outcome that will influence the planning of the next dive.

Validation of the underlying models

Some years ago, factors related to bends-rates were analysed, in confidence, for the UK Department of Energy by Shields.[38] This project was dependent on the North Sea contractors providing the commercially sensitive paper records (logs) for each of many thousands of air dives. Verification of the data and elimination of some erroneous dive records from the analysis were conducted in accordance with predetermined criteria. They also provided their bends data which was reviewed by an independent and experienced consultant. Figure 2 (on page 210) shows two values of Shield's index of decompression severity ($P\sqrt{t}$) plotted over a background of diving activity in the North Sea.[38]

On the public release of the results there was concern that the basis for this analysis, the use of $P\sqrt{t}$ as a measure of potential decompression severity, was not valid for the subsequent decisions relating to decompression performance.[6] $P\sqrt{t}$ was used as an index of only the gas loading at the end of the dive's bottom time, assumed that the dive had been spent entirely at its maximum recorded depth, and so took no account of the decompression, which of the available commercial sets of decompression tables had been used, nor what additional safety factors of depth and duration had been introduced when the specific decompression profile for the actual depth and duration was selected, nor how well the actual decompression had been followed. For example, none of the contractors followed the published USN Surface Decompression tables but, because of bends experience, each contractor had introduced their own private extensions to the final oxygen decompression from 40 feet (12 m) to the surface. If that did not work maybe they modified them again, and so every table was different. Nor did $P\sqrt{t}$ reflect deviations from the established maximum of a 5 minute surface interval in the surface decompression procedures.

Within the wide range of the different "proven" tables selected (but probably no "cowboy" tables) and the range of ways in which they are actually used, it is not surprising that it was said that there are no obvious differences between them in their gross effectiveness, as estimated on the basis of classifying all dives just by depth and bottom time. Because of commercial confidentiality, the HSE was not able to publish comparisons between company tables but has said informally that there were no detectable differences.

Thus it seems that all these different tables were so similar that any differences in bends rates between them were swamped by the other factors, such as the use of hot-water suits. Nevertheless, in spite of those deficiencies, which were due to decisions taken by the Department of Energy (later the Health and Safety Executive), the results of their recommendations were surprisingly effective and were based on the observation that distribution of

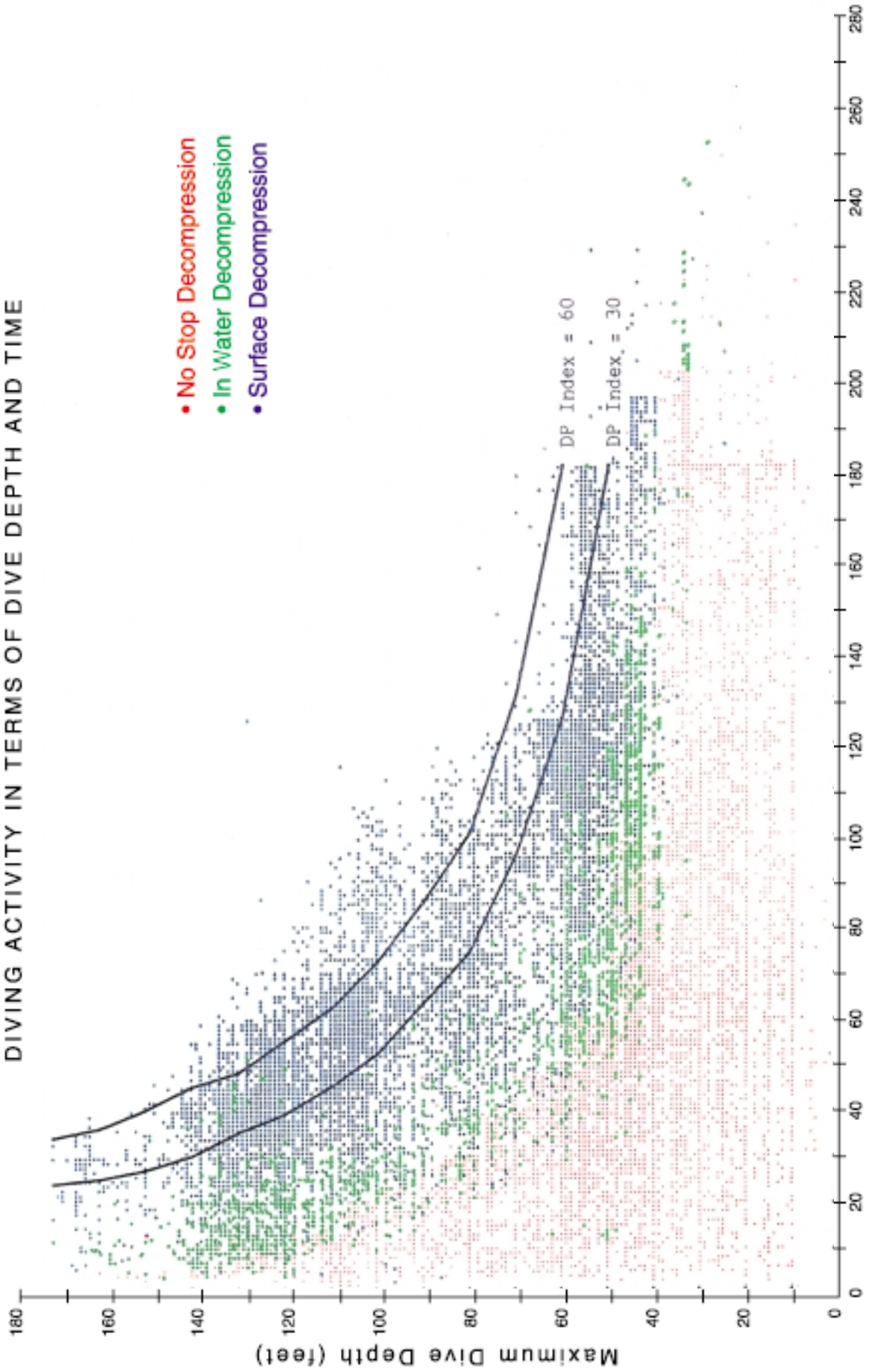

Figure 2. Two values of Shield's index of decompression severity (plotted over a background of diving activity in the North Sea. No stop dives are red dots, in water decompression dives are in green dots and surface decompresssion dives are blue dots.

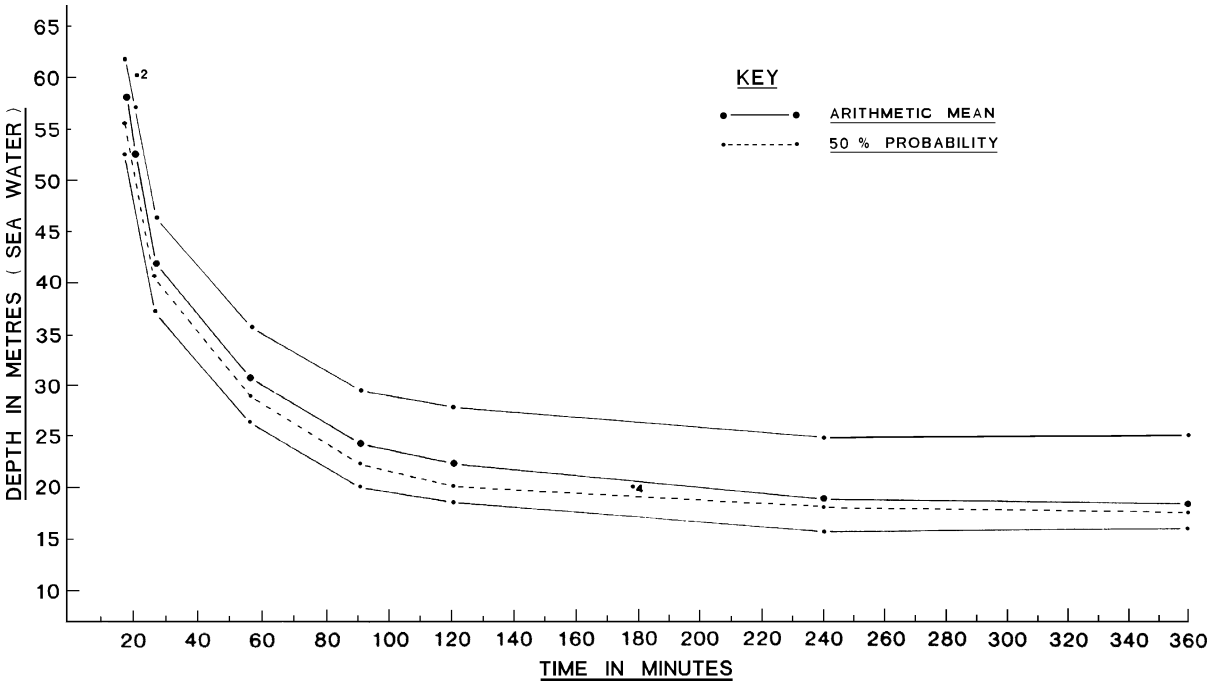

Figure 1. Hempleman's study of the wide variability of no-stop curves (mild bends end point) in goats. Below the bottom line no bends would be expected. Between the bottom and top lines only mild bends were recorded and it can also be seen that the distribution within these limits is not symmetrical but skewed. Above the top line severe bends would be expected. *Reprinted with permission from The Physiology and Medicine of Diving, 4th edition. Bennett PB and Elliott DH. Eds. London: Saunders, 1993; 355 [Fig. 13.5].*

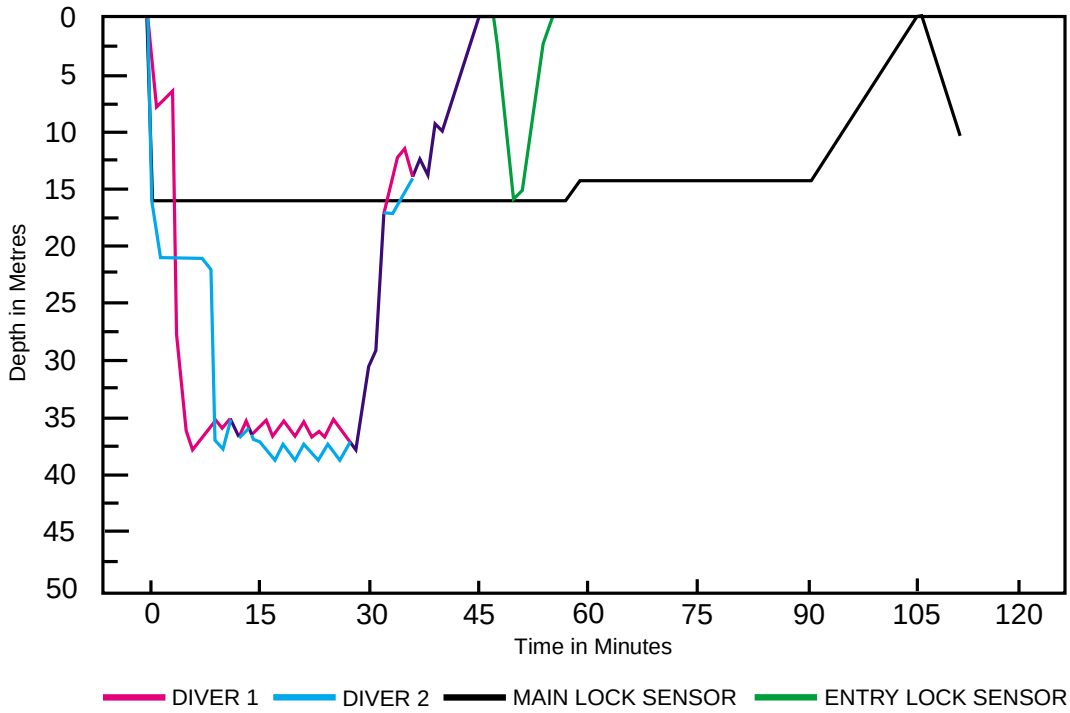

Figure 3. An illustration of depth-time recording. Diver Blue descends but waits until his buddy, Diver Red, has not only cleared his ears but has made it to the bottom before he continues down. These plots would be from on-line depth recorders which can be watched in real time by the surface supervisor. After a fairly erratic in-water stop they proceed to surface where the surface decompression chamber (black) is already at some 16 metres. The divers descend to it in the outer lock (green) which returns to surface after the divers have transferred to the main compartment and, after moving to 14 metres to complete their 40-min stop, they finally return to the surface.

decompression illness was predominantly related to the P√t index of dive severity. So, for air and nitrox diving, the UK Department of Energy issued a Safety Memorandum in 1988 introducing a "limiting line" and it has since been reissued by the UK Health and Safety Executive and adopted in the industry's Approved Codes of Practice.[39] This restriction is effective in reducing the bends rate simply by declaring dive exposures beyond the limit as possible only with a special HSE dispensation. The imposition of this limit on the diving industry brought the annual recompression rate for all air-range dives in the North Sea down to below 0.04% and, for two of these years at least, to zero.

Since then personal on-line depth-time recorders have been introduced for working dives in the North Sea. The recording of the depth and time at frequent intervals during the course of many actual dives and their decompressions are currently being collected, on-line from hose divers.[40] The acquired profile is detailed and even the effects of waves upon maintaining the depth of an in-water stop are capable of being quantified. Figure 3 (on page 211) is an illustration of depth/time recording of a dive with surface decompression following the depths achieved and the pressures in the two compartment chamber. The computerised records of these dives, together with other relevant details of each dive such as the gas breathed and the bends outcome, are to be available for central analysis, probably by the statistical method of maximum likelihood, in order to modify any inconsistencies within the diverse decompression models used for the generation of both tables and personal computer profiles. From the experience gained by this feedback, improvement of the underlying models may enable divers to approach the limiting line of the tables more safely, and then to venture beyond the present boundary of relatively safe dives towards the deeper and longer dives tables that retain the greater decompression risk unacceptable to industry. Paradoxically the future of the dive data recording study on working divers in the North Sea is threatened because there are "not enough bends", to the extent that it could take several decades to complete just a pilot study.

The future of this approach to validating the underlying decompression models lies with monitoring dives with potentially greater decompression risks. Studies on recreational divers, with post-dive down-loading from personal data loggers, are underway and will provide data from many thousands of actual dives which will be analysed to improve decompression safety.[41,42]

Personal decompression safety

However far the highly-refined decompression models of the future will enable divers to penetrate beyond the present limits of relative safety, none of this progress can change the fact of biological variation. Adaptation to decompression stress or the opposite, susceptibility, will still be unpredictable for the individual's next dive. Incorporating the user's physiological characteristics into a personalised decompression model would not control these or any other contributory factors on every dive. The use of Doppler also has practical limitations, not least because the detection of bubbles does not mean the onset of symptoms. The ultimate objective is some way of monitoring on-line the individual's potential during a dive for the development of the pathological effects of bubbles later.

But the story of that will have to be in some future historical account of decompression safety.

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STRATEGIES FOR TREATING DECOMPRESSION SICKNESS.

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Key Words
Decompression illness, physiology, treatment,

Introduction

Decompression has generally been regarded as safe as long as it does not lead to clinical symptoms requiring treatment. Traditionally, the symptoms following decompression (dysbarism) has been distinguished according to where the main symptoms occur (Table 1).

This classification implies that the different categories are well defined disease entities and that there is reasonable agreement between doctors about the classification. Both the study of Smith et al.[1] and a study by Kemper et al.[2] demonstrate that there is considerable uncertainty between experts about classification. For instance, cerebral DCS cannot, in many cases, be distinguished from arterial gas embolism or vestibular barotrauma. Furthermore, several studies have shown that symptoms only from joints are quite rare, they are usually accompanied by central nervous symptoms,[3,4] Extreme fatigue can be classified as a harmless sign or be a sign of subclinical pulmonary embolism.[5] Francis et al.[6] therefore suggested the term decompression illness to include both decompression sickness and arterial gas embolism. They furthermore suggested that the disease should not classified as Type I and type II, but instead described according to clinical symptoms and their development. Using this classification scheme, a high degree of concordance between different doctors was reached .[7]

TABLE 1

CLASSIFICATION OF DECOMPRESSION DISORDERS (DYSBARISM)

Decompression sickness

Type I (mild)	Type II (serious)
Muscles and/or joints (bends, niggles)	Spinal
Skin	Cerebral
Lymph	Vestibular
Malaise/Fatigue ?	Cardiopulmonary (Chokes)

Arterial gas embolism
Barotrauma

Clinical diagnosis and reporting

"The major symptoms and signs of decompression sickness are pain (bends), asphyxia (chokes) and paralysis. Minor effects are rash and fatigue. The parts of the body chiefly involved are the extremities (bends), cardiorespiratory system (chokes) and the spinal cord".[8]

Even today, there is probably little to add to this description by Behnke in 1951, with the possible exception that we believe today that the brain may be more frequently involved and that extreme fatigue may be a more serious sign than previously thought.[5] However, it must be borne in mind that the symptoms can be slight and, as was described by one author, "as many as in syphilis and diabetes together".

In decompression disorders, the patients have to report their symptoms before treatment or investigations can be initiated. In many cases, the patients do not report their symptoms, either because they do not recognize them as being related to the dive or they feel reluctant to do so for many reasons.

There has been, for many years, anecdotal evidence that clinical symptoms of DCI are underreported to a considerable degree. We have recently asked a large group of Norwegian divers about this.[9] 19% of the sports divers, 50 % of the professional air divers and 63% of the saturation divers reported that they had symptoms that had not been treated, a majority of these symptoms were related to the CNS. Interestingly enough, there was a statistical relationship between this and later minor central nervous symptoms.

The incidence of decompression sickness.

There is probably little argument that severe violation of decompression procedures will lead to serious

symptoms and that these are caused by widespread gas bubble formation in many different organs. However, decompression illness requiring treatment is a rare disease. In commercial diving, the incidence of treated DCI is probably below 0.1%.[10] In recreational divers, the incidence is probably considerably below this. However, these general numbers hide the fact that some types of dives, even in commercial operations, have a much higher incidence of DCI. This was seen in the study by Shields and Lee,[11] where the majority of the incidents happened in the more stressful dives, as defined by a high $p\sqrt{t}$, where p (pressure) is the maximum depth of the dive in bar and t is the duration of the dive in minutes.

Even if decompression illness is quite rare, a large percentage of divers have been treated. In a survey among divers in an off-shore diving company in 1985, 38% of the divers with 1-9 years experience and 62% of those with 10-24 years of experience had been treated for decompression sickness.[12] A recent survey of a large population of Norwegian divers, showed that 3% of the recreational divers and 28% of the experienced professional divers had received treatment during their career.[13]

Table 2 shows an overview of symptoms of decompression sickness in several studies over a time period of 90 years.

Even given the possibility that there may be differences in reporting, there are remarkable differences in the symptomatology. Of particular interest is to note that pain is only present in about half of the cases in the amateur divers. Furthermore, that serious injuries of the spine and symptoms from the lungs are quite common in amateur divers. This might fit in with the observation that 17% of the amateurs had experienced extreme fatigue. This sign has been described as a sign of subclinical pulmonary embolism.[5] According to Lehner et al.[17] shallow and long or deep and short dives have a high incidence of chokes. The latter dives also have a high incidence of central nervous DCI. The main difference between these dives are the tissues that will be supersaturated. Thus, the change in symptomatology might indicate a different diving practice and that the decompression procedures are not adequate for the more stressful dives.

Pathophysiology of decompression illness

There seems to be no disagreement that the basic problem in decompression illness is the formation of gas bubbles in the organism. The studies of Boycott et al.[18] which form the basis of most decompression procedures, used the concept of allowable supersaturation, indicating that there was a level of supersaturation that could be tolerated without problems occurring. Many studies since then has shown that this level of allowable supersaturation is actually only related to clinical symptoms, not to bubble formation. Any supersaturation can lead to bubble formation. While many studies confirm this, there is a remarkable difference in the actual occurrence of bubbles between individuals and in one single individual at different times. We do not know the reason for this, but believe that there are significant differences in the number of nuclei present. These nuclei may be composed of small (approx. 1 micron) stable gas bubbles.[19] Furthermore, it is well known that the stress of the dive, including temperature and physical work, can increase the number of bubbles observed. It is important to be aware of the fact that the effect of environmental factors will be greater on the least stressful dives.

Gas bubbles have only been observed in a few locations, even after experimental and very stressful dives.

TABLE 2

INCIDENCE OF SYMPTOMS IN DCI

Subjects	**Caisson workers**	**US Navy divers**	**US Navy divers**	**Recreational divers**	**Recreational divers**	**Occupational divers**
Authors	**Keays**	**Behnke**	**Rivera**	**Kidd**	**DAN**	**Kelleher**
Year	1909 [13]	1947 [8]	1964 [14]	1969 [15]	1993 [16]	1994 [4]
Number	3,692	159	935		1.249	225
	%	%	%	%	%	%
Pain	89	72	92	70	57	67
Rash		14	15		4	5
Paralysis	0.9	0.6	6		6	
Fatigue			1		17	13
Visual disturbances		5	7		6	4
Chokes/Dyspnoea	1.6	4	2		9	8

These are the fat around the viscera, the white matter (myelin sheets) in the central nervous system, in the blood and in the fascia and capsules around joints.[20] There is little reason to doubt that the localized pain in a joint is caused by local gas formation. This has been elegantly demonstrated by Webb,[21] who showed that gas could be seen in periarticular and perivascular tissue spaces and that there was a correlation between the occurrence of gas and pain. Ferris and Engels further demonstrated that strain and muscular activity were correlated with pain at the site where the strain had been applied.[22] One further observations would tend to support this, namely the fact that local compression can in many cases remove the pain. Ferris and Engels claim that the pain can be eliminated by eliminating arterial inflow.

There is evidence from many studies that gas bubbles occur in the venous system during most decompressions.[23-26] Data from the study of Eckenhoff et al.[27] indicate that once the sum of the partial pressures of all gases exceeds the environmental pressure, gas formation occurs in the venous system.

Generally, the main focus on the lungs has been on its role as a filter, where the bubbles are eliminated before they can be transmitted to the arterial side, where their potential for damage is greater. However, if the gas load on the lungs is large, the filtering capabilities of the lungs will be exceeded and gas will enter the arterial circulation.[28] Furthermore, if an patent foramen ovale (PFO) is present, as it is in about 25-30% of the younger population,[29] gas bubbles will be transmitted to the arterial side at much lower pressures.

Several studies have documented the relationship between the occurrence of many venous bubbles and the risk for clinical symptoms requiring treatment.[23,30,31] This, together with the fact that bubbles probably are present in the venous system during most decompressions, suggests that a diver complaining of pain in a joint may be suffering from two different conditions, namely tissue gas in and around the joint and pulmonary gas embolism.

It has been suggested that there is little relationship between gas bubbles detected in the pulmonary artery and clinical signs of decompression illness (DCI). The main reason for this is that gas bubbles have been detected in the absence of symptoms.[24] There seems, however, to be agreement that the risk of DCI increases with increasing number of bubbles. In our experience, having monitored many hundreds of air dives and numerous saturation dives, clinical symptoms do not occur in the absence of pulmonary artery gas bubbles. Nishi points out that for air dives, decompression illness was always accompanied by vascular bubbles.[24] Sawatzky[32] has shown that there is a 5-10% risk of decompression illness in individuals with a single observation of grade III - IV bubbles, using the grading system developed by Spencer and Johanson.[30]

Bubble formation is only the initial insult. The surface of the bubbles act as a foreign substance and will initiate numerous biochemical processes. In vitro studies have demonstrated that gas bubbles have an effect upon both formed elements and biochemical processes in the body. Using gas bubbles in vitro, Thorsen et al.[33] showed that gas bubbles lead to aggregation of thrombocytes.

Ward et al[34] demonstrated that gas bubbles could activate complement in-vitro. Using a different technique, Bergh et al.[3] were able to verify this. The importance of this mechanism in-vivo is still unclear. However, responses of the endothelium to gas bubbles seems to be important in decompression sickness. Chrysanteou et al. have shown that animals exposed to decompression will show breakage of the blood- brain-barrier.[36] Broman et al. have demonstrated that even very short contact between gas bubbles and endothelium (1-2 minutes) will lead to such breakage.[37] Furthermore, studies in rabbits indicate that such contact leads to endothelial damage and progressive reduction on cerebral blood flow and function.[38]

In the central nervous system, bubbles seem to form both in the vessels and in the myelin tissue.[39] Experimentally, it has been shown that after short, deep dives a significant number of individuals have significant hemorrhages in the spinal cord, these individuals are very refractory to treatment.[40]

Generally, decompression illness is considered mostly a "bubble disease". Even if bubbles most probably are the initiating event and the sometimes dramatic response to pressure increase show that the mechanical effect of bubbles certainly play a role, the biochemical reaction to the bubble surface must be considered. This leads to endothelial damage, aggregation of cells on the endothelial surface and an inflammatory response.[41,42] If bradykinin, a strong vasodilator that requires an intact endothelium for its effect,[43] is given after decompression, a dramatic increase in mortality occurs.[44] If however, the animals are treated with anti-inflammatory drugs before the dive, mortality and histological changes are significantly reduced.[44]

Is decompression illness a disease?

According to Webster's dictionary[45] a disease is:

"a condition of an organ, part, structure or system of the body in which there is incorrect function resulting from the effect of heredity, infection, diet or environment. A disease is a serious, active, prolonged and deep-rooted condition."

In contrast to this

"A disorder is usually a physical or mental derangement, frequently a slight or transitory one."

I think there is probably no disagreement when we say that DCI is potentially a disease if it is not treated properly, I will also claim that it can be a disorder if proper action is taken. The aim of all our effort must be to keep DCI as a disorder.

Even if acute clinical symptoms are not present, organic changes may occur. A recent consensus conference determined that such changes, even in individuals with few or no reported symptoms, have been found in the bones, central nervous system and the lungs.[46] The changes are, however, small and probably of little functional significance. They seem to by quite well documented for the lungs and much less defined for the central nervous system. A study of the spinal cord of 20 experienced divers, several with bone necrosis and with a history of decompression sickness, showed absolutely no changes.[47]

This certainly raises the question of how to regard vascular gas bubbles (as detected by ultrasound) without any clinical symptoms, the so-called "silent bubbles" described by Behnke.[8] Most will probably not regard this as DCI. However, the fact that such bubbles are present during most decompressions is similar to the situation in many infectious diseases with detectable pathological flora and few or no symptoms. The question still remains whether these bubbles can have an effect on the organism. We have recently been able to show in the pig that the degree of endothelial damage in the pulmonary artery is dependent upon the number of gas bubbles, if few bubbles were present (less than Grade III on the Spencer scale) no damage could be found.[48]

Initial treatment of DCI

The basis for any treatment is a correct diagnosis. As is pointed out above, this is not easy. Furthermore, many of the treatments are initiated by individuals with little medical and clinical training. If the diver is treated immediately after the onset of symptoms, then treatment is mostly successful and the particular procedure used is probably not very important. However, there is no clear definition of prompt treatment, even a delay of a few hours may reduce the chances of full resolution. If treatment is not prompt, the treatment results are usually less favourable, with residual clinical symptoms being seen in about 50% of the cases.[4] This is particularly the case when there is a long delay between injury and treatment, if this is more than 12 hours, about 70% of the individuals have residual symptoms.[49]

The initial treatment, if a pressure chamber is not available, is breathing oxygen. DAN data, both from Europe and the USA, has shown that this significantly reduces clinical symptoms and reduces the number of sequelae. Preferably a demand valve should be used with a well fitting mask. In more severe cases, fluid may be given i.v. The successful use of other drugs, even if theoretically advantageous, has not been documented.

The basis of any definite treatment of decompression illness is pressure and oxygen. There are four effects of this treatment.

- Increase in environmental pressure. This will reduce the size of the gas bubble and thus reduce the risk of ischemic damage.
- Increase in oxygen partial pressure in blood and tissue. This will increase the gradient for inert gas removal.
- Increase in the oxygen content of arterial blood. This will increase the oxygenation of the tissue, thus reducing the risk of hypoxic damage.
- Biochemical and reactive effects of oxygen. These effects, although the least understood, may be highly significant in the treatment of DCI.

The treatment of decompression sickness has till now been based on mostly empirical data, where a standard treatment has been applied to every case of decompression sickness. The only exception to this has been that in some serious cases has one tried treatments using higher pressures, other gases or saturation. There has been no clear criteria for choosing one over the other.

Recommended treatment pressures vary from 200 to 780 kPa, while oxygen tensions vary from 220 to 300 kPa. However, as was pointed out in a recent workshop,[50] compression to 18 msw (280 kPa) breathing 100% oxygen is the only procedure where extensive clinical experience exist. This treatment should therefore probably be the basic treatment in all cases. In most cases this means the use of USN Table 6. However, several studies have documented that both shorter tables at the same depth[51] as well as treatments at 200 kPa[52] give equally good results. Recently, this last group published that 70% of the divers with neurological symptoms were symptom free after two to six hours at 200 kPa and that 13% of these divers had persistent manifestations after one month.[53]

There is very little data to support higher treatment pressures. However, most people with experience in the field have case histories where a patient that show no improvement at 280 kPa improved on reaching 600 kPa either breathing air or a nitrogen/oxygen mix. Treatment at 600 kPa used to be the recommended treatment for air embolism. The theoretical basis for this is that an increase in pressure will reduce bubble size. However, the reduction in bubble size is the largest at the first doubling of pressure (100 - 200 kPa). Indeed, Gorman et al. showed in the rabbits that the vascular bubbles in the brain were cleared as effectively using 202 kPa as using pressures up to 1010 kPa.[54] This is also supported by the study of Kunkle and Beckman,[55] who showed that bubble resolution time would decrease by a factor of two if oxygen at 280 kPa was

compared to the use of oxygen at surface and that further increase in pressure would not decrease this time further.

During recent years, there has been considerable discussion about the use of helium / oxygen mixtures, mostly the use of 50/50 heliox at 400 kPa (COMEX 30). This procedure was developed by a diving company who claim to have excellent results with this approach. There are three difference between this approach and the USN Table 6, namely a higher environmental pressure (400 vs 280 kPa), a reduced oxygen tension (150 vs 280 kPa) and the use of helium.

Some animal studies performed by Hyldegaard[56-58] seem to support the use of heliox over 100% oxygen, in particular if the bubbles are located in fatty tissue. However, the advantage of using helium is largely lost if helium is introduced at 280 kPa.[59] A further problem is the location of the bubbles. If they are located in the white matter of the brain, which contains only about 20% fat and where the elimination of the bubbles is largely diffusion limited, a nitrogen bubble will grow. We have demonstrated the growth of such bubbles in aqueous gels for over a week.[60]

The increased pressure may be of benefit. We have shown in pigs that the gas bubbles in the pulmonary artery disappear significantly quicker when recompression is performed according to Comex 30 compared to USN Table 6.[61] Recent extensions of this study demonstrate that bubbles in the pulmonary artery disappear at the same rate for compression pressures from 200 - 400 kPa.[62] In performing these studies, we were impressed by the effectiveness of recompression to 200 kPa even using air. Animals with a large number of gas bubbles, with hardly any heart beat and no respiration recovered immediately on arrival at pressure. This would be a strong support for recompression even in divers who are terminally ill.

The use of lower oxygen tensions may actually also be of benefit. Leitch and Hallenbeck showed that oxygen at 200 kPa was the optimal treatment gas in spinal cord decompression sickness in dogs.[63]

The dose of oxygen has only been considered to a limited degree when evaluating treatment procedures. In general, there is a belief that more oxygen is better and that the only limitation is oxygen toxicity. Oxygen is a vasoconstrictor, at oxygen tensions of about 200-280 kPa, blood flow to all organs will be reduced by approximately 20-25%.[64] Furthermore, as oxygen tensions increase, the shunt fraction in the lung will increase, thus reducing the effect of higher oxygen tensions.[65]

More importantly, oxygen at pressure has numerous biochemical effects which may be of importance when judging the optimal dose of oxygen. If indeed vascular obstruction and endothelial damage plays an important role in decompression illness, decompression illness may be compared to reperfusion injury. Blocking leukocyte adhesion[66] and C5a activation[67] by monoclonal antibodies significantly reduce the injury after ischemia and reperfusion. In these situations reactive oxygen species play a significant role[68] and it is reasonable to assume that the correct dose of oxygen is important for successful treatment. For example, it has been demonstrated that the glucose metabolism in the injured brain improve after 35-40 minutes at 150 kPa oxygen, but deteriorated after 15 minutes exposed to 200 kPa.[69] Timing of treatment as well as the tissue at risk probably also plays a role.

Thom et al. have shown that a single 45 minute exposure to an oxygen tension of 280 kPa will completely block activation of leucocytes, a mechanism of central importance in tissue injury and endothelial damage, this effect lasts up to 8-10 hours.[70]

The use of drugs is at present largely experimental and no definite recommendations can be made. In France, aspirin is regularly used,[53] although in-vitro studies have demonstrated that acetylsalicylic acid has no effect on platelet aggregation induced by gas bubbles.[71] If further studies demonstrate that endothelial damage plays a significant role, this will open exciting possibilities for drug treatment.

An adequate circulatory volume should be maintained, but it is important to keep in mind that overhydration may lead to the risk of cerebral edema, particularly if the blood-brain barrier has been damaged. If fluids are used, it is important to keep in mind that there is growing evidence that hyperglycaemia will significantly increase the injury of the central nervous system.[72] If treatment is performed in a hot climate it is also important to be aware of the fact that hyperthermia also will lead to an increased injury.

Treatments if initial treatments are not successful

The definition of a non-successful treatment is not easy. In many cases, the patient improves under pressure, but some symptoms are still present. This can be handled either by keeping the patient under pressure and performing more oxygen cycles or by going deeper using some of the options available or by decompressing to the surface and performing follow up treatments. There is not enough data to support any one approach. Data from DAN indicates that more than 5 follow-up treatments may not give any additional benefit.[73]

The future

There is still much that is unknown in the treatment of decompression illness. One particularly interesting point

is the question whether a standard treatment should be used for all cases of decompression illness. Due to the difference in the speed of uptake and elimination of gas in the different tissues, it is likely that the gas load in the different tissues, and thus the degree of bubble formation, will be different in different tissues. A short, deep dive will produce bubbles in quite different tissues from a long shallow dive. This is in accordance with what was pointed out by Lehner, that different dives produce different symptomatology.[74] Central nervous symptoms are more common in deep, short dives, while long, shallow dives produce predominantly symptoms from joints and muscle. Computer simulations support this and also indicates that bubbles from such dives disappear more quickly using pressures at 400 kPa with 50% oxygen than when using USN Table 6.[57] Thus an approach like the one used by Comex for many years may actually have considerable merit, where they treat minor symptoms at 220 kPa and go to 400kPa for more serious symptoms.

At present we do not have sufficient information to make adequate decisions about optimal treatment strategies. Such information is urgently needed, considering the large number of individuals who are left with sequelae after treatment with today's procedures. This is even more important as we can expect new challenges as recreational divers will be able to go deeper, stay longer and use a number of gas mixes.

A possible future strategy for treatment

1 Oxygen as soon as possible after the insult.

2 Initial treatment regardless of symptoms apply pressure and oxygen, 200 kPa ?

3 If not immediate response of treatment or more than 2 hours between insult and treatment, evaluation of case:

what is the gas load in different tissues ?
where are the bubbles located ?
are there many bubbles in vessels ?
are there bubbles in the brain / spinal cord tissue ?
will the resolution of the bubbles be dependent upon diffusion or perfusion limitation ?
what role does an inflammatory responses play ?
is hypoxia an important part of the picture ?

4 The evaluation above shall result in a treatment algorithm that will define optimal pressure, breathing gas and decompression procedure as well as the optimal use of drugs.

It must be pointed out that much of this information is not available today. It can, however, give us a framework for what information may be of importance and the need for the development of the necessary methods for obtaining that information.

Acknowledgment

This work has been supported by Philips Petroleum Norway as part of the HADES project.

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SPUMS ANNUAL SCIENTIFIC MEETING 1997

RECOMPRESSION THERAPY FOR DECOMPRESSION ILLNESS

Richard Moon

Key Words

Decompression illness, oxygen, treatment.

Abstract

Recompression therapy for decompression illness was developed empirically based on observations by compressed air workers. The rationale that was developed fit the evidence that the disease was caused by bubbles, and it has been presumed that the major mechanism of action is related to physical reduction of bubble size. Oxygen was later added to increase the gradient for diffusion of nitrogen from bubbles, and to relieve tissue hypoxia. Definitive treatment of decompression illness (DCI) includes the administration of oxygen under pressure. Current recommendations include initial recompression to 2.8 bar, using USN, RN or closely related commercial procedures. A review of experimental data and experience with recompression tables is discussed. Expeditious application of recompression using oxygen along with standard resuscitative measures is usually successful in treating decompression illness. Recent evidence suggests that pharmacological effects of hyperbaric oxygen, in addition to the physical effects on bubble size, gas diffusion and oxygenation, may be important in resolving the disease.

Introduction

Recompression therapy dates back to the 19th century. The bridge across the Mississippi River at St. Louis, completed in 1874, at was an engineering milestone in the United States, because the bottom of the Mississippi is covered in mud and it was impossible until that time to construct piers using traditional bridge building techniques. In order to excavate down to bedrock the engineers used what resembles an upside-down cup (caisson), into which was pumped compressed air to maintain the internal pressure equal to that of the hydrostatic pressure outside. As the caisson rested on the bottom, the air pressure prevented the ingress of water and mud, allowing workers inside to facilitate pumping of the mud to the surface. The caisson gradually sank by its own weight, aided by the mass of the bridge pier being constructed atop the caisson. Once on bedrock, the caisson was filled with concrete, locking the bridge pier permanently into place. This was the first major use of caisson construction work in the United States.

At the end of a shift the men decompressed in an independently pressurised lock. As the depth (and hence the ambient pressure in the caisson) increased, the men were subjected to progressively increasing decompression stress. Many of the men developed neurological decompression sickness (DCS) and 14 of them died. It is perhaps of note that as a result the engineer, James Eads, hired a local doctor, Dr Alphonse Jaminet, who then became the first occupational physician in the United States concerned with the welfare of men working under pressure, to take care of the men. This man, although not knowing the pathophysiology of decompression sickness, elucidated several procedures and principles for the prevention of this illness that are still believed correct to this day.[1]

One of Dr Jaminet's contributions is an account of an episode of spinal cord bends that he experienced after leaving the caisson following a visit to the work site. With no definitive treatment available, other than tincture of time, he went home, drank some wine and gradually got better. Unfortunately this was not the fate of Washington Roebling, the engineer of the Brooklyn Bridge, built a few years afterward using the same technology, who became permanently disabled by spinal cord DCS after helping to fight a fire in the caisson.

It was noted that when men with DCS re-entered the caisson on their next shift they felt better and their symptoms often resolved. This was the beginning of recompression treatment, which has continued to be developed to this day. The use of recompression therapy was not routine, however, until the East River tunnels in New York City were constructed between 1906 and 1909, and described by Dr. Frederick Keays.[2] Keays observed that 89% of bends cases treated with one or two recompressions experienced complete relief, compared with only 75% of cases treated "by medical means". He also observed the relationship between delay to treatment and residual symptoms. Eight percent of cases treated within less than two hours were left with residual symptoms, vs. 25% of cases treated 12 or more hours after symptom onset. The history of the development of recompression tables has been lucidly described by Chris Acott.[3]

TABLE 1

AMBIENT PRESSURE AND PO_2 OF RECOMPRESSION TABLES

Table	Maximum pressure (bar)	PO_2 at maximum pressure (bar)
Comex Table 12[27,45]	2.2	2.2
USN Tables 5, 6[19]	2.8	2.8
Comex Table 30[27]	4	2
USN Table 6A[19]	6	1.2
Modified USN Table 6A[17]	6	3
USN Table 8[19]	7.8	1.4

Recompression treatment

It is now understood that bubbles are the initiating cause of decompression illness, and therefore it is logical to believe that increasing the ambient pressure, and so reducing the bubble size, will be therapeutic. Boyle's Law predicts that bubble volume is inversely proportional to the ambient pressure. If the bubble is spherical, the reduction in volume will be accompanied by a less impressive reduction in diameter. At 2.8 bar (18 m), a commonly used pressure for the treatment of decompression illness, the immediate reduction in bubble diameter is only about 30%.

Another effect of recompression with oxygen is the relief of tissue hypoxia and oedema. Recently in animal models of arterial gas embolism,[4] ischaemic myocutaneous flap[5] and carbon monoxide poisoning[6-8] there has been evidence that leucocyte adherence to damaged endothelium plays a pathophysiological role. Furthermore, in humans hyperbaric oxygen administration reduces leucocyte adherence by inhibiting beta-2 integrin function.[9] This is likely to be another beneficial effect of recompression treatment for DCI.

Over the years there has been considerable discussion about the optimum depth and PO_2. Recompression chamber operations are usually limited to 50 m (6 bar); inspired PO_2 is usually within the range 2.5-3 bar, above which there is an unacceptable risk of toxicity. A wide range of treatment depths and inspired PO_2 values has been published (Table 1).

One of the first studies to address the issue of treatment depth was performed by Waite, who injected air into the carotid arteries of dogs and, using a cranial window to observe bubbles directly, studied the resolution of these bubbles as a function of ambient pressure.[10] He found that at a chamber pressure of 4 bar (30 m) all of the bubbles originally visible in the cranial window had disappeared. The logical suggestion that the maximum pressure needed to treat arterial gas embolism should be 4 bar was not implemented by the US Navy because it was felt that fleet diving medical officers would not accept any table with a maximum depth less than 6 bar. Since then, Des Gorman and colleagues, using a rabbit model, have examined a range of pressures and made two interesting observations. First, bubbles often passed directly through the vascular network without becoming trapped, even without recompression. Second, bubbles that ended up in communicating vessels, through which there was very little blood flow, did not resolve, even after recompression to 6-10 bar.[11]

Other studies have examined the effect of recompression on brain electrical function. In one series of experiments, Leitch and colleagues examined the effect of various ambient pressures on in dogs with gas embolism. Somatosensory evoked response amplitude was used as an end point, and no differences in efficacy among ambient pressures between 2.8 to 10 bar were observed.[12] McDermott, in two feline studies published in 1992, examined recompression to 6 bar on air followed by 2.8 bar on oxygen versus 2.8 bar on oxygen without deeper recompression, and found no differences,[13] nor were there were differences when an enriched O_2 mixture was administered at 6 bar.[13]

Similar lack of effect of pressures greater than 3 bar has been observed in a canine model of spinal cord decompression sickness.[14] No advantage to recompressing to 7 bar breathing air was observed.

Table 2 illustrates the partial pressures of the different component gases in inspired and alveolar gas, arterial blood, tissue, and bubble. At 1 bar breathing air here is a slight difference in partial pressure of nitrogen from the bubble to the tissue of about 150 mm Hg. For this reason a tissue bubble eventually resorbs because the inherent diffusion gradient facilitates gas diffusion from the

TABLE 2

PARTIAL PRESSURES OF NITROGEN IN ALVEOLAR GAS AND BUBBLE.

The calculated partial pressure gradient from bubble into surrounding tissue, assuming tissue PN_2 = alveolar PN_2, is shown.

Pressure (bar)	FiO_2	Alveolar PN_2 (mmHg)	Bubble PN_2 (mmHg)	Bubble PN_2-Tissue PN_2 * (mmHg)
1	0.21	571	713	142
1	1.00	0	713	713
2.8	0.21	1664	2096	432
2.8	1.00	0	2096	2096

* For this calculation it is assumed that tissue PN_2 = alveolar PN_2.

The inherent difference between bubble and tissue PN_2, which increases during O_2 breathing, is known as the "oxygen window".

bubble to surrounding tissue, from where it is transported away by the blood.

After recompression to 18 m (2.8 bar), in addition to reduction in bubble volume, the partial pressures of oxygen and nitrogen are raised, and the N_2 diffusion gradient increases from 150 to about 440 mm Hg. However a disadvantage is that the increased partial pressure of nitrogen loads up other tissues with inert gas, which subsequently has to be washed out.

Breathing oxygen at the surface (1 bar) also increases the nitrogen diffusion gradient, from about to about 700 mm Hg, illustrating one advantage of breathing oxygen at the surface compared with recompression breathing air. Recompression to 18 m (2.8 bar) breathing 100% oxygen raises the gradient for diffusion to over 2,000 mm Hg. This is the basis for modern treatment of decompression illness: recompression while breathing supplemental O_2.

The question of the optimal PO2 has been examined in several animal studies. Leitch and colleagues, using a canine model of spinal cord DCS in which the animals were recompressed to 5 bar, observed that the optimal PO2 was between 2 and 2.5 bar.[15] However the investigators used somatosensory evoked responses to monitor recovery, which in our experience in human DCI, do not correlate well with clinical response. The optimum PO_2 for humans has not been established, but clinical experience suggests that it is in the range 2.5-3 bar.

The conclusion of these experiments on the treatment of DCI is that if pressure and increased partial pressure of oxygen are administered, the differences among different pressures and PO_2 values are fairly small, at least in animal studies using direct observation of bubbles or electrophysiological function as measures of outcome.

Which table to use

Although a wide range of pressures and PO_2 values are used with current treatment tables (Table 1, page 225), the most commonly used recompression table is US Navy Table 6 (Figure 1). It consists of administration of oxygen and air cycles at 18 m (2.8 bar, 60 ft), then decompression, breathing oxygen, to 9 m (30 ft) over 30 minutes, then a number of cycles of oxygen and air, followed by a 30 minute decompression, breathing oxygen, to the surface.

It is very flexible, as the number of cycles can be extended at both 18 and 9 m. US Navy Table 5 (Figure 2, page 225), designed for pain only bends, is a shortened version, which consists of only two cycles of oxygen at 18 m 60 feet and a reduction in the time at 9 m. US Navy Table 6A is a Table 6 preceded by 30 minutes at 50 m (6 bar, 165 ft) breathing air (Figure 3, page 226). It was initially recommended as initial treatment for arterial gas embolism. A subsequent modification of it by civilian practitioners substitutes 40-50% O2 for air at 50 m,[16,17] but today it is rarely used.

The Table 6 two-step "paradigm" has been taken perhaps to its ultimate limit in the Catalina Marine Science Center Treatment Table, which appeared in the SPUMS Journal 12 years ago.[18] Divers taken to the chamber on Catalina Island had to be shuttled between the hospital on the mainland in Los Angeles and the chamber on the Island. This table was therefore designed to administer as much oxygen to the diver during the initial treatment as practically feasible. The Catalina Treatment Table is shown in Figure 4 (page 227).

Current US Navy[19] and recreational[20] guidelines recommend (for treatments initiated from the surface) an initial treatment depth of 18 m, with deeper recompression

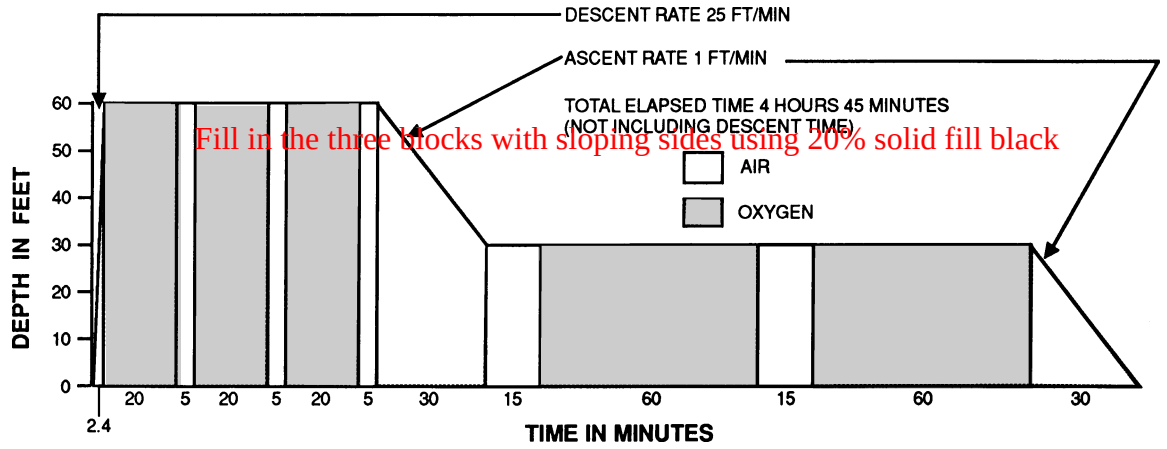


Figure 1. USN Treatment Table 6. Table 6 is used for treatment of neurological decompression illness and patients with pain-only or mild cutaneous symptoms that are not relieved within 10 minutes of reaching 60 ft breathing oxygen. Table 6 can be extended at 60 ft and at 30 ft (9 m, 1.91 bar) if symptoms have not been relieved within the first three oxygen cycles. (Drawing reproduced from Moon[55]).

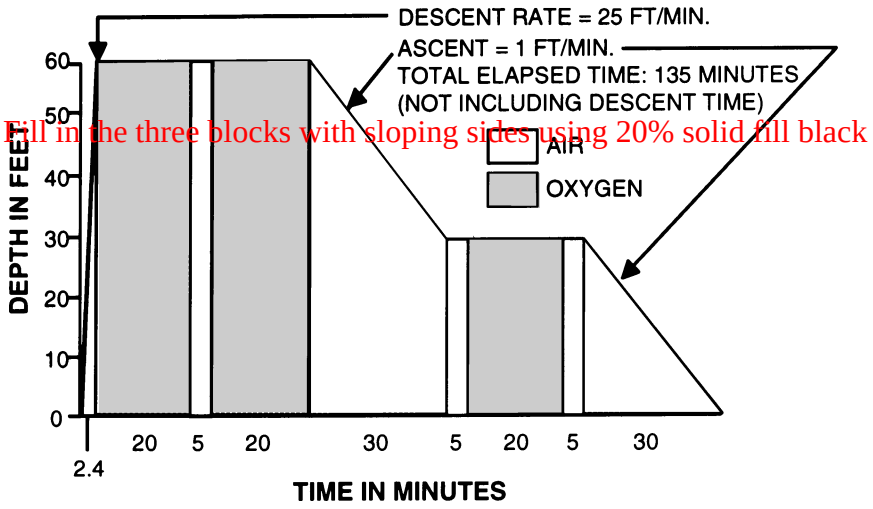


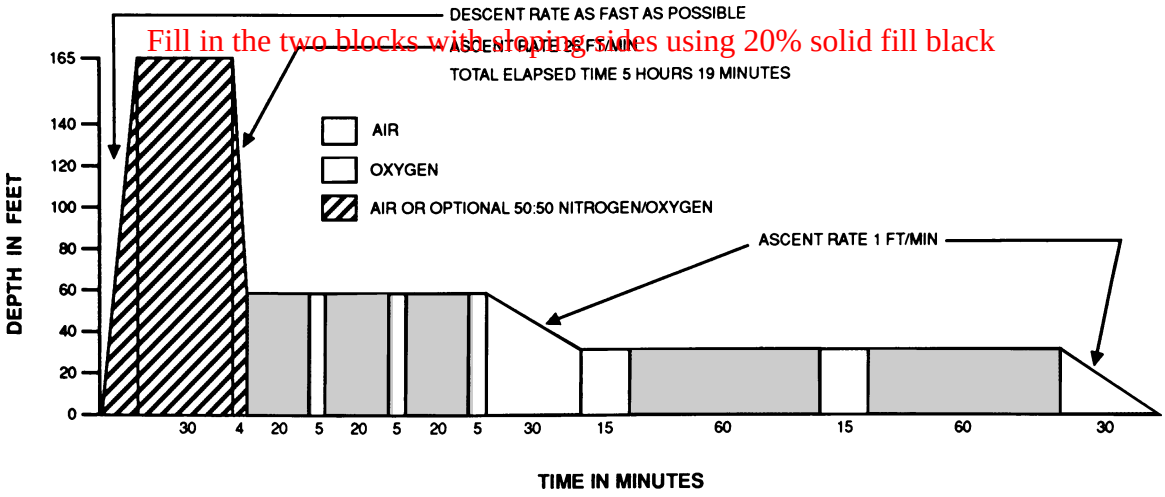
Figure 2. USN Treatment Table 5. This treatment table is recommended for pain-only or mild cutaneous symptoms with no neurological symptoms or signs. If complete relief of symptoms has not occurred within 10 minutes of compressing the patient to 60 ft (18 m, 2.8 bar), then Table 6 is recommended. (Drawing reproduced from Moon [55]).

as an option available for instances of inadequate clinical response. Indeed, the vast majority of cases of decompression illness will respond satisfactorily to one or more applications of an 18 m treatment table.

Table 3 (page 228) shows a number of published series, largely from military experience, of divers with DCI including a total of 1,763 patients. After the first treatment around 80% had complete relief. Most ultimately attained asymptomatic status after one or more extra treatments. Objective evidence indicates the high degree of success of these treatment tables in military practice, and supports their wide acceptance. However, in recreational diving, where it is rare to have immediate access to a chamber, the success rate is less than optimal. In Ball's series[21] (Table 3) the success rate in civilian divers was poor, seemingly attributable to treatment delay. In recreational divers in the Divers Alert Network (DAN) database delay to treatment is common. Of 483 cases of DCI reported in the 1998 DAN Report only 17% received recompression therapy within 4 hours of symptom onset, and 43% within 12 hours.[22] Delay to treatment is a factor that is associated with poor outcome.[23]

Shallow or short recompression

Short tables, such as those designed for use in monoplace chambers, also appear to be effective.[24,25] The monoplace table designed by Hart specifies 100% oxygen



Fill in the two blocks with sloping sides using 20% solid fill black

Figure 3. USN Treatment Table 6A. This table consists of a 30 minute excursion to 6 bar (50 m, 165 ft) while the patient breathes air, followed by an oxygen breathing portion identical to USN Table 6, and was initially recommended for treatment of arterial gas embolism. While the U.S. Navy still recommends air as the appropriate breathing gas at 6 bar, others have used nitrogen-oxygen mixtures (usually 60:40 or 50:50). Some animal studies have indicated that compression beyond 2.8 bar provides no additional benefit. Nevertheless, in clinical experience a small percentage of patients may respond to treatment at 6 bar, but fail to do so at 2.8 bar. (Drawing reproduced from Moon[55]).

administration at 3 bar for 30 minutes followed by 2.5 bar for 60 minutes.[24,26] These shorter tables appear to be effective in most cases, although they have not been prospectively compared with the more commonly used schedules such as USN Table 6, and their equivalence to the longer oxygen tables in severe decompression illness is questionable.[25]

Data presented by Imbert[27] suggest that for pain only bends a 12 m (40 ft; 2.2 bar) recompression for two hours is adequate. In his retrospective review of DCI treatments in commercial divers he reported 91% success in 407 cases. Few details are provided, however, and whether this treatment modality can be applied to recreational divers, in whom long delays to treatment are common, is uncertain.

Deeper recompression

There are several published tables incorporating initial recompression to depths exceeding 18 m. Comex Treatment Table 30,[27] for example, incorporates an initial recompression to 30 m for 60 minutes, breathing either 50-50 N_2-O_2 or He-O_2 (see Figure 5, page 228); USN Treatment Table 6A was described earlier. A modification of Table 6A has been described by Lee and colleagues,[17] who reported a series of divers who had very severe disease, with long delays to treatment. In their modified treatment tables, the divers spent 60 minutes at 50 m (6 bar, 165 ft) breathing 40% oxygen and were then decompressed according to Table 6A. The published results were remarkable: 70% of 99 divers were cured and 29% were improved. It was not a randomised trial, however, and it is possible that improvements attributed to the modified table were due to general supportive procedures such as fluid resuscitation.

To examine the issue of deeper recompression after an initial period at 18 m (2.8 bar), in 1985 Leitch and Green retrospectively reviewed a number of cases of DCI in naval divers. Fourteen of their cases were recompressed to 50 m (6 bar) breathing air. Six cases were cured, however two were already improving at 2.8 bar and only one had motor abnormalities. Five cases had marginal improvement, and in three cases there was no effect. Compression beyond 6 bar was implemented in 10 cases, in whom there were two cures in divers with sensory problems only, and 8 instances of incomplete or no improvement. Four of these 8 relapsed during decompression. Their retrospective data review did not suggest that this was hugely successful. Recompression to greater than 2.8 bar or greater than 18 m should be an available option, the information that is in the literature suggests that it is rarely useful.

Figure 6 (page 229) shows data from 3,899 cases of decompression illness reported to DAN, illustrating the relationship between probability of complete relief and delay to recompression. The probability of complete relief is greater if recompression is administered early rather than late. However, even divers treated after 12 hours or more delay have some relief of symptoms. Response to recompression treatment of DCI, even after several days' delay, has been reported.[28-31]

Recently there has been some interest in using helium as a component of a treatment gas for divers with

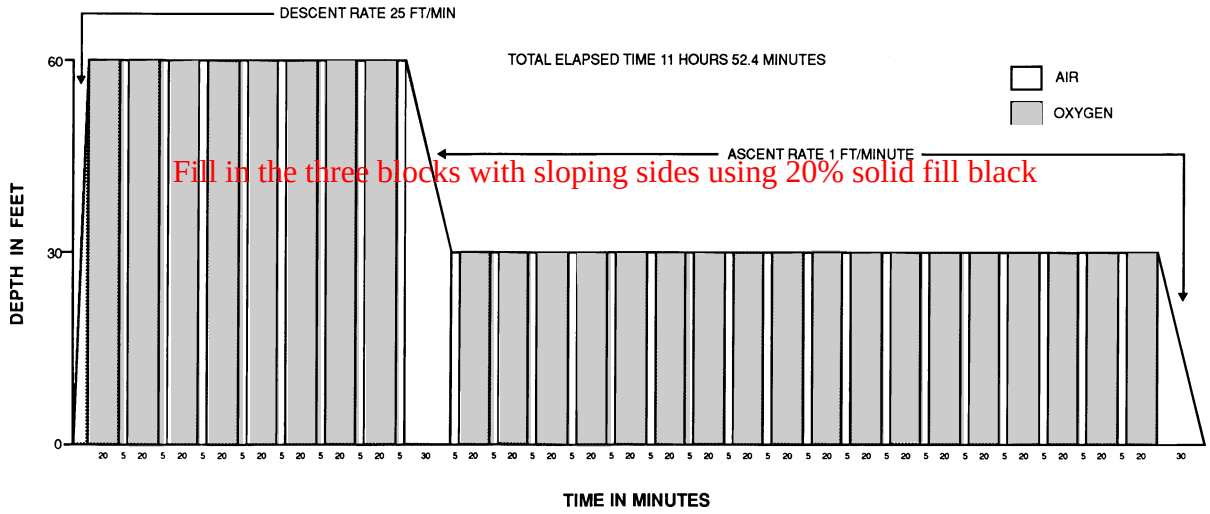


Figure 4. Catalina Treatment Table. This is a modified version of USN Treatment Table 6. All oxygen breathing cycles are of 20 minutes duration, followed by 5 minutes breathing air. In its current implementation, shorter versions of this table may be used as follows. After 3 oxygen cycles at 60 ft, a minimum of 6 cycles are required at 30 ft (equivalent to USN Table 6); after 4 cycles at 60 ft, 9 cycles are required at 30 ft; after 5 to 8 cycles at 60 ft, a minimum of 12 cycles are required at 30 ft. Up to 18 cycles at 30 ft (as shown above) can be used. Tenders must breathe oxygen for 60 minutes at 30 ft and during the decompression to the surface (total 90 minutes). If there have been fewer than 4 oxygen cycles at 60 ft and fewer than 9 cycles at 30 ft, then only 30 minutes of oxygen breathing is required for the tender at 30 ft in addition to the decompression time (total 60 minutes). Further treatments can only be started after 12 hours of air breathing at the surface. Further details of this treatment table have been published by Pilmanis. (Drawing reproduced from Moon[55]).

DCI after air or nitrox dives. There is some reason to believe that recompression with helium might offer some advantage. The movement of a gas through a liquid depends upon its solubility in the liquid and its diffusivity, a function of molecular weight. The rate of flux of gas along a partial pressure gradient is related to the partial pressure difference and the gas permeability: the product of solubility and diffusivity. The permeability of helium in oil is less than that of nitrogen. Therefore, in fatty tissues it might be possible that breathing helium-O_2 could allow nitrogen to diffuse out of a bubble faster than helium diffuses in. Indeed, in a study by Hildegaard and colleagues, when bubble size in rat spinal cords at 1 bar was measured as a function of time, the rate of bubble diameter shrinkage was faster when the animals were administered He-O_2 than when they received 100% O_2.[32] In another rat experiment, breathing He-O_2 prevented the development of spinal cord DCS after a chamber air dive to 3.8 bar for one hour, and appeared to be superior to 100% O_2.[33] It is also conceivable that there are pharmacological effects of helium unrelated to gas diffusion. In an animal study, using tissue oxygen electrodes in the cerebral cortex, cortical PO_2 was highest when the animals breathed He-O_2 compared with air or 100% O_2 at the same inspired PO_2.[34]

Two small uncontrolled series of human DCI suggested that He-O_2 recompression is effective.[35,36] Imbert reported the results of Comex Table 30 (Figure 4). He found when using nitrox during the 60 minute period at 30 m, four out of 25 divers required an additional treatment, whereas when heliox was used none of 11 required further treatment.[27] These observations are consistent with an advantage of heliox, although the difference between the two treatments is not statistically significant.

However, in a guinea pig model of severe DCI Lillo and colleagues[37] observed that recompression with He-O_2 resulted in a slower recovery from tachypnoea than when air was used as the treatment gas. A retrospective review of cases treated in the US Navy indicated no advantage of tables using He-O_2 vs. those using N_2-O_2/O_2.[38]

A randomised trial[39] currently (1997) underway in Auckland may decide the issue. In the meantime there is no compelling reason to switch from using O_2 treatment tables to those incorporating He-O_2.

How many treatments ?

Most diving physicians recommend repetitive treatment with hyperbaric oxygen until the patient's symptoms have resolved, or until there is no further improvement after a treatment (clinical plateau). The vast majority of cases of DCI will respond to a single recompression treatment. Although a small minority of divers with severe neurological injury may not reach a clinical plateau until 15-20 repetitive treatments have been administered, formal statistical analysis of approximately 3,000 DCI cases in the DAN database supports the efficacy of no more than 5-10 repetitive treatments for most injured

TABLE 3

SINGLE RECOMPRESSION SUCCESS RATE OF USN OXYGEN TREATMENT TABLES
(from Thalmann[38])

Source	Number of cases	Complete relief (%)	Substantial relief (%)	Comments
Workman[46]	150	85	95.3 (after 2nd treatment)	
Erde & Edmonds[47]	106	81		
Davis[48]	145	98		Altitude DCS
Bayne[49]	50	98		
Pearson & Leitch[50]	28	67	83	
Kizer[51]	157	58	83	Long delays
Yap[52]	58	50	84	Mean delay 48h
Gray53	812	81	94	
Green[54]	208	96		All pain only, USN Table 5
Ball[21]	14	93 (mild cases)		{
	11	36 (moderate cases)		{ Many cases with long delays
	24	8 (severe cases)		{
TOTAL	**1763**	**81**		

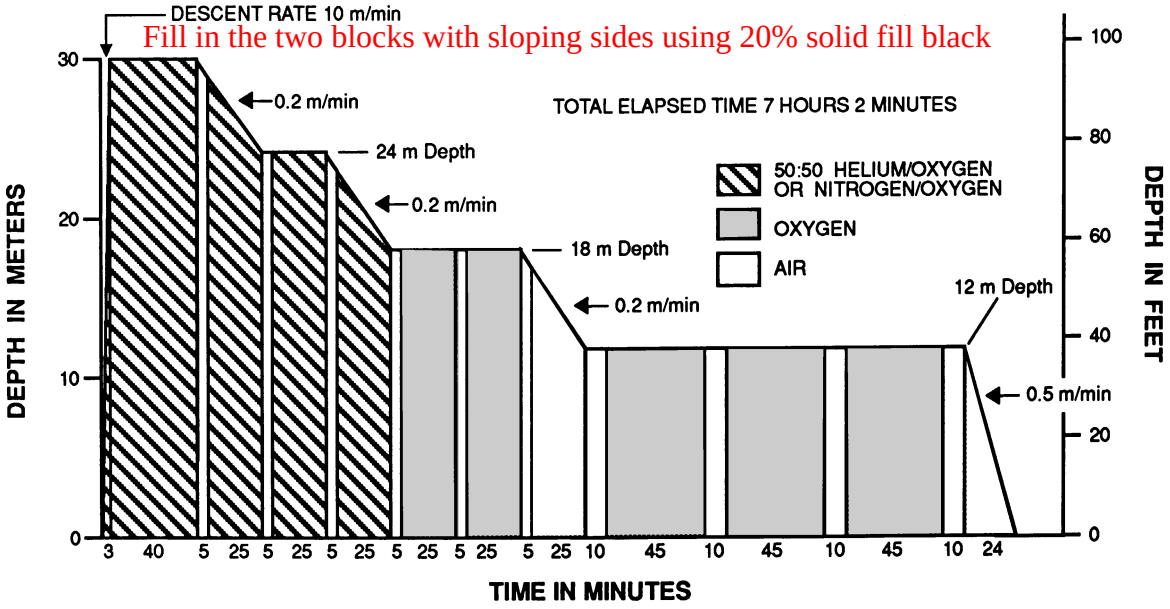


Figure 5. Comex Treatment Table 30. This table is an option for the treatment of DCI, and can be implemented using either N_2-O_2 or He-O_2 for the period at 30 msw (approximately 100 ft). In European practice, this table is frequently implemented as the initial treatment of decompression illness. (Drawing reproduced from Moon[55]).

divers.[40-42] Symptoms and signs that are unresolved at the end of hyperbaric treatment usually continue to resolve spontaneously for months or even years.

There are few data that address the issue of which treatment table to use for follow-up treatment. A retrospective review by Wilson and colleagues[43] suggested that follow-up tables at 18 msw (2.8 bar, 60 ft) were associated with a lower relapse rate than those at 14 msw (2.4 bar, 46 ft). The analysis was subject to the potential pitfalls of a retrospective review, and pending the availability of more definitive data, there is no incontrovertible basis upon which to recommend any particular follow-up table.

Saturation treatment

Saturation recompression treatment is a technique in which the chamber remains for a prolonged period at treatment pressure until the patient's symptoms are resolved or maximally improved. Saturation treatment can be

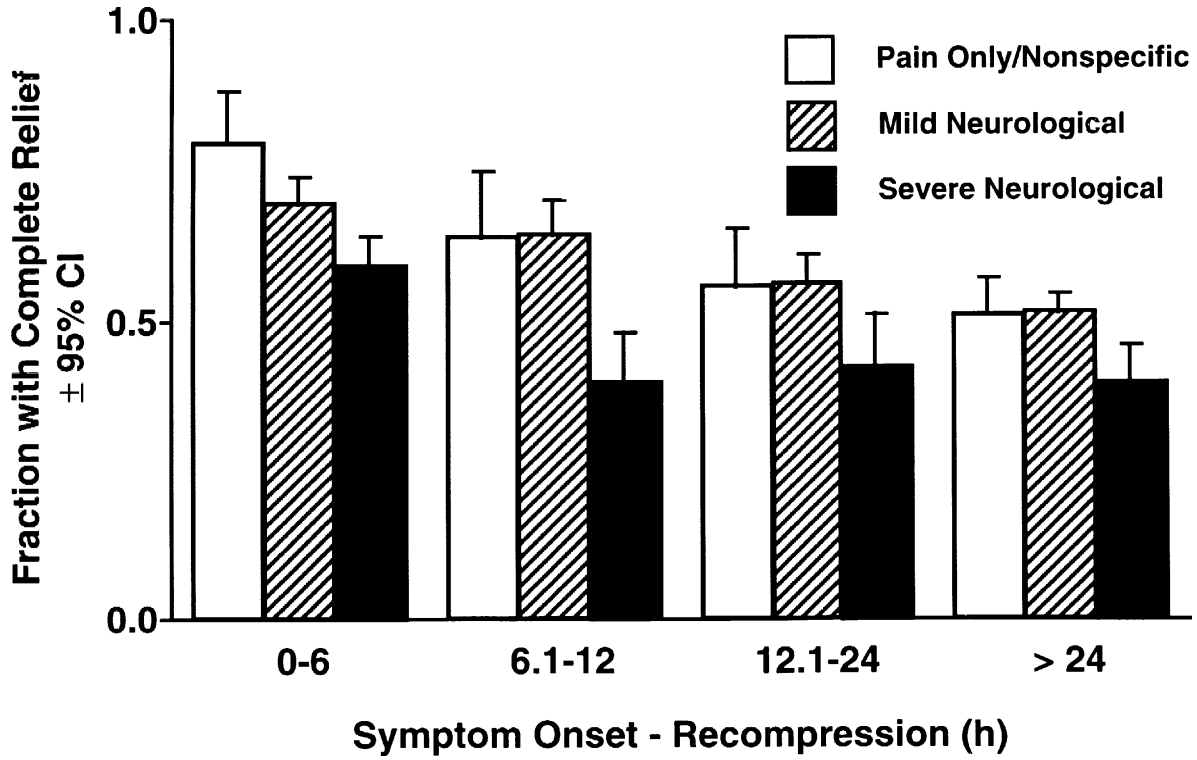


Figure 6. Residual Symptoms after Recompression Treatment. Data represent 3,899 cases of DCI in recreational divers reported to DAN, and are stratified by severity: Severe neurological symptoms include convulsions or abnormalities of vision, gait, urinary/anal sphincter function, motor strength or consciousness.

routinely implemented in commercial diving practice, and has been used occasionally in the treatment of recreational diving casualties.[16] It requires a chamber in which the atmospheric carbon dioxide and oxygen can be accurately monitored and controlled. Saturation treatment optimally requires at least two tenders and a chamber large enough to care for a critically ill person comfortably. Treatment depth is typically 18-30 m (2.8-4 bar); depths exceeding 18 m require reduction of ambient PO_2 to maintain a mixture that is not toxic (PO_2 typically 0.4-0.6 bar). USN Treatment Table 7 is one of the easiest saturation treatment tables[19] The saturation depth for this table is 2.8 bar (18 m, 60 ft), and hence air can safely be used for the chamber atmosphere.

The Duke University experience with saturation treatment in 16 divers since 1977 has been reported previously.[44] The 15 divers who failed an initial USN Table 6 treatment were compared with a similar group of 10 who subsequently received multiple short O_2 treatments (USN treatment tables followed by twice daily treatments at 2 bar for 120 minutes). Following treatment, gait tended to be better in the divers treated with saturation tables. One week post accident 5 of 15 divers who received saturation treatment could walk with or without assistance, vs. none of 10 in the short O_2 table group. At hospital discharge one third of the divers treated with saturation tables could walk independently, vs. only one in the short O_2 table treatment group.

Indications for considering saturation treatment are severe neurological DCI, and either continued improvement at 18 msw even after a maximum number of oxygen cycles has been administered, or significant deterioration during decompression.

Summary

Definitive treatment for DCI is recompression using an oxygen enriched breathing mixture. Treatment protocols (“tables”) have been empirically designed, and using an initial treatment pressure and PO2 of 2.5-3 bar have a high degree of success. Use of a standard treatment table is recommended, with follow-up treatments administered until resolution or clinical plateau. Treatment pressures in excess of 3 bar are rarely required. Advantages of administration of a breathing gas other than O_2 or N_2-O_2 (e.g. He-O_2) have not yet been substantiated.

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THE VASCULAR ENDOTHELIUM CURRENT CONCEPTS OF CIRCULATORY HOMEOSTASIS AND PATHOPHYSIOLOGY.

Paul Langton

Key Words

Cardiovascular, physiology.

Abstract

The vascular endothelium is increasingly recognised as an active tissue, with key roles in the maintenance of a non-thrombogenic, semi-permeable circulatory barrier, variable vascular tone, and the regulation of platelet reactions and leucocyte trafficking. Central to these functions are the endothelial derived mediators of nitric oxide, prostanoids and the cell adhesion molecules.

Nitric oxide is produced by healthy endothelium and acts as a flow dependent vasodilator, an inhibitor of platelet aggregation and a regulator of CAM production. Prostacyclin, the principal endothelial prostanoid, is both vasodilatory and a potent anti-platelet agent. The cell adhesion molecules include selectins, integrins, and members of the immunoglobulin superfamily. They participate in crucial cell-cell and cell-matrix interactions such as platelet activation, leucocyte adhesion and migration of cells to sites of inflammation.

The dependence of a wide variety of disease states, from myocardial infarction to decompression illness, on these reactions opens up new therapeutic potential for intervention with novel agents.

ABBREVIATIONS TABLE

5HT	5hydroxytryptophan
AA	arachadonic acid
ACE	angiotensin converting enzyme
ACEI	ACE inhibitors
ADP	adenosine diphosphate
Ang II	angiotensin II
ATIII	anti-thrombin III
CAM	cell adhesion molecules
COX	cyclo-oxygenase
CPB	cardiopulmonary bypass
ECE	endothelin converting enzyme
EDHF	endothelium dependent hyperpolarising factor
EDRF	endothelium derived relaxing factor
eNOS	endothelial nitric oxide synthetase
ET-1	endothelin
IFNg	interferon gamma
IL1	interleukin 1
IL8	interleukin-8
NO	nitric oxide
NOS	nitric oxide synthetase
nNOS	neuronal nitric oxide synthetase
PG	prostaglandin
PGI_2	prostacyclin
SAH	subarachnoid haemorrhage
TF	tissue factor
TNF	tumour necrosis factor
TxA2	thromboxane
vWF	von Willebrand factor

Introduction

Over the last decade our understanding of the endothelium has evolved from that of a simple lining of the vascular tree into one of a specialised tissue with a range of complex functions. The interrelation of these often opposing functions at a molecular level is only now starting to be unravelled. This overview aims to highlight some of the recent advances in endothelial function that are particularly relevant to general and underwater medicine.

Vascular Tone

ENDOTHELIAL VASODILATORS

Vascular tone has traditionally been viewed as balance of direct acting, sympathetic and para-sympathetic, constrictive and dilatory effects on vascular smooth muscle. Local factors such as temperature, pH and PCO_2 regulate minute to minute blood flow to specific tissues. Furchgott was the first to demonstrate that vasodilators could have indirect actions, via the endothelium, on vascular tone.[1] It is now well recognised that the potent vasodilator, endothelium derived relaxing factor (EDRF) is responsible for the effects of many intrinsic and exogenous vasodilators (fig 1).

EDRF has subsequently been identified as the ultra-short lived product, nitric oxide (NO), and/or more persistent thiol-adducts (proteinaceous complexes of NO with sulphur containing amino acid residues). NO is produced from L-arginine through the action of the nitric oxide synthetase (NOS) family of enzymes. NO is released into the vessel lumen where it exerts anti-platelet effects, and into the vessel wall, producing vasodilatation. Nitric oxide synthetase is always present on healthy endothelial surfaces (eNOS) and possibly also in the bronchial epithelium. Its activity is increased by shear stress and by oestrogen. Many intrinsic (bradykinin, histamine, acetylcholine, serotonin) and extrinsic vasodilators (various drugs) act to increase eNOS activity and NO generation. Therapeutic nitrates, in the form of glyceryl trinitrate, and isosorbide mono- (Imdur) and di-nitrates (Isordil) are metabolised by the endothelium to release NO, whereas nitroprusside, a direct NO donor is an endothelium independent vasodilator. Both thrombin and endothelin

increase eNOS activity, which may serve to counteract the vasoconstrictive properties of these agents.

The first pathophysiological role ascribed to NO was in subarachnoid haemorrhage (SAH). Free intracranial haemoglobin is a potent inactivator of NO and contributes to the development of vasospasm in SAH. Volume expansion in this setting increases shear stress and so NO production. The diffuse vasospasm of SAH may be (in part) analogous to that seen in neurological decompression illness (DCI).

Large amounts of NO can be produced by inflammatory cells in response to stimulatory cytokines (eg tumour necrosis factor and interferon g). NO toxicity contributes to the adverse negative inotropism, peripheral vasodilatation and pulmonary congestion of septic shock. Indeed, persistent exposure of many cell types to high levels of NO (e.g. in septicaemia) leads to their apoptosis (programmed cell death).

A second potent endothelial derived vasodilator is prostacyclin (PGI_2), which is produced mainly in small arterioles and capillaries. Prostanoids are derived from arachadonic acid (AA), by the actions of cyclo-oxygenase (COX) and then specific prostaglandin (PG), prostacyclin and thromboxane (TxA_2) synthetases. PGI_2 may have only a minor vaso-active role in health but is an important anti-platelet agent. PGI_2 is increased in diabetics, and contributes to their higher resting limb blood flow (independent of any autonomic neuropathy) compared to non-diabetics. However in diabetes, complex endothelial dysfunction leads to an overall impairment of normal exercise induced vasodilatation, largely due to impaired NO production.

The third endothelial vasodilator was initially described on the basis of functional "endothelium dependant vaso-relaxation", not inhibited by a combination of NOS and COX blockers and which was associated with hyperpolarised arterial smooth muscle. This substance has been termed endothelium dependent hyperpolarising factor (EDHF) or endocannabinoid, as its vascular effects are mediated by binding to intrinsic cannabinoid receptors. EDHF contributes significantly to vasodilatation of intra-myocardial arteries, and generation of EDHF is thought to contribute, in part to the coronary dilation of bradykinin.

ENDOTHELIAL VASOCONSTRICTORS

The most potent vasoconstrictor identified to date is the polypeptide endothelin (ET-1). ET-1 is formed from the sequential enzymatic cleavage of a larger precursor protein. The final step is catalysed by endothelin converting enzyme (ECE), in a manner analogous to the angiotensin converting enzyme (ACE) dependent synthesis of angiotensin II (Ang II) (fig 1). Adrenalin, Ang II, chronic hypoxia (e.g. high altitude), and thrombin increase the

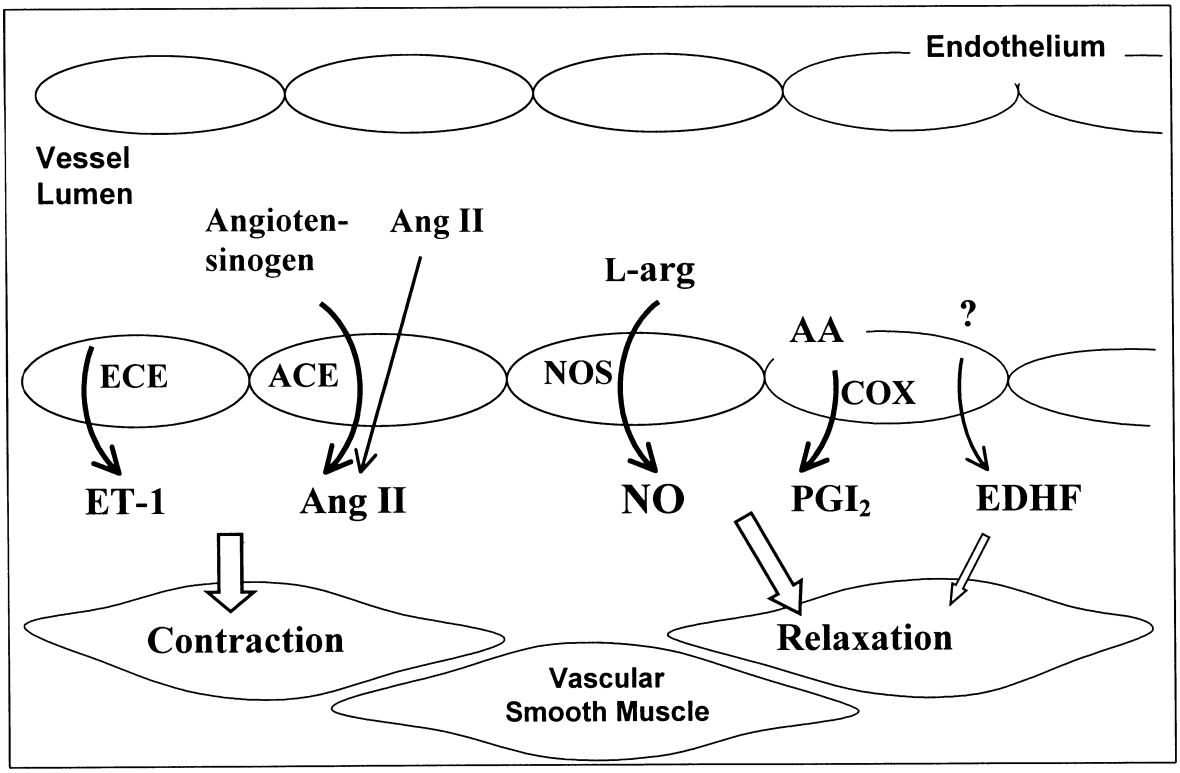

Figure 1. Maintenance of vascular tone by a balance of vasodilating and vasoconstricting substances. For abbreviations, refer to abbreviations table.

production of ECE and hence ET-1 production. Circulatory levels of ET-1 are very low, suggesting it acts mainly locally within a few mm of where it is produced. Its vasoconstriction is more potent on veins, intramyocardial-coronary and pulmonary arteries. ET-1 acts on ET_A receptors on vascular smooth muscle to produce vasoconstriction and on endothelial ET_B receptors leading to NO and PGI_2 production. The first clinically relevant ET receptor antagonist, bosentan, is undergoing phase III trials for the treatment of heart failure and primary pulmonary hypertension.[2]

The renin-angiotensin system is an important regulator of both vascular tone and Na+/K+ balance. Local tissue ACE and Ang II generation is known to be important in regulating regional blood flow and angiogenesis, as well as in myocardial remodelling after infarction. Ang II vasoconstricts via a direct action on smooth muscle angiotensin receptors and by increasing local ET-1 production. ACE inhibitors (ACEI) causes vasodilatation by inhibiting these mechanisms, by enhancing renal Na+ excretion, and by reducing the breakdown of bradykinin. The benefits of ACEI are limited alternative mechanisms of Ang II generation, involving tissue chymases and other enzymes. This has lead to the development of specific AT_1 receptor antagonists, including irbesartan (AvaproÆ), which has been released in the Australasian market place. It is likely that this new therapeutic class will have synergistic effects with ACEI.

The vasoconstricting effects of the prostanoids TxA_2 and prostaglandin H2 (PGH_2) are relevant to local responses to endothelial damage and platelet plug formation. They are otherwise not considered to play a major role in homeostasis of vascular tone.

Haemostasis : Platelets and Thrombosis

Normal haemostasis is a complex interplay between the vessel wall, platelets and the coagulation and fibrinolytic systems.

In the absence of vascular disruption a non-thrombogenic, semi-permeable, endothelial barrier must be maintained. This is achieved in part by high local concentrations of PGI_2 and NO to which platelets are exposed during their capillary transit. The anti-platelet effect is sufficient to ensure that most platelets remain inactivated under normal conditions. In all but the smallest blood vessels, normal laminar flow prevents the formed elements of blood from coming into contact with the endothelium. Additionally, intact endothelium produces thrombomodulin, heparan sulphates (Heparans are a similar but distinct group of native glycose-amino-glycan substances to the broad group of substances known as therapeutic heparin.) and anti-thrombin III (ATIII) proteins, which inactivate thrombin and stimulate the fibrinolytic system. The endothelium is also a potent source of ADPase, the enzyme that breaks down adenosine diphosphate (ADP) which is produced by platelets.

After endothelial damage, blood elements are exposed to collagens, tissue factor (TF) and von Willebrand factor (vWF) in the subendothelial matrix. An initial platelet monolayer is formed in response to adhesion of their surface glycoprotein-Ib receptors to tissue vWF. Platelet degranulation (the "release reaction") ensues with 5hydroxytryptophan (5HT) and ADP excretion leading to further recruitment of platelets. The final step of platelet activation is the production of glycoprotein-IIb/IIIa, which allow fibrinogen binding and further platelet accumulation. The multilayered "platelet plug" so formed provides initial haemostasis (Fig 2). Subsequent "fluid phase" haemostasis is dependent on activation of the coagulation system. This is enhanced by the production of TF on the surface of activated platelets and endothelial cells. TF production is increased by hypoxia and by inflammatory cytokines such as interferon gamma (IFNg) interleukin 1 (IL1) and tumour necrosis factor (TNF). Thrombin is generated by the coagulation factor Xa and leads to fibrin cross linking (with stabilisation of the platelet plug), further platelet activation, release of various vasoactive endothelial substances and stimulation of fibrinolytic pathways.

The events contributing to thrombosis are endothelial damage with platelet activation, stimulation of the coagulation cascade, reduced local PGI_2 and NO formation producing vasospasm, loss of laminar flow, stasis and further accumulation of activated platelets and coagulation factors.

Leucocyte trafficking and migration

Trafficking is the term used for the two-way movement of leucocytes between the circulation and tissues, both during normal cell recirculation, and during migration into areas of tissue injury or inflammation. The ability of leucocytes to be rapidly recruited to areas of tissue injury is dependent on the variable production and activation of specific cell adhesion molecules (CAM) on both the endothelium and leucocytes. The three major families of CAM involved in regulation of leucocyte migration are the selectins, the integrins and the immunoglobulin superfamily (Table 1). These facilitate the sequence of leucocyte rolling, activation, tight adherence and tissue migration (fig 3, page 236).

Endothelial cells have low levels of both E- and P-selectin on their luminal surface which are rapidly increased in response to local tissue injury. Endothelial selectins interact reversibly with L-selectin on the surface of leucocytes, slowing leucocyte flow in a process termed "rolling". This facilitates subsequent leucocyte activation by local cytokines and enables cell-cell interactions that

would otherwise be prevented by normal vascular shear rates.

The integrins consists of a series of a and b-chain heterodimers, which are sub-classified according to the b-chain. Leucocyte migration principally involves the b_1 (CD29) and b_2 (CD 18) families. On leucocytes, surface production of CD 11/18 is markedly increased when cells are activated by complement, leukotriene B4, TNF or the interleukin-8 (IL8) family of chemokines. The b_3 integrin, gp IIb/IIIa, forms the principal adhesion molecule of activated platelets.

The adhesion molecules ICAM (1,2 & 3), VCAM and PECAM (CD 31) belong to the immunoglobulin superfamily. Endothelial ICAM and VCAM are the counter receptors for b_2 and b_1 integrins respectively. Normal endothelial ICAM production is low, but increases rapidly upon endothelial activation. After the initial selectin mediated rolling phase, the formation of ICAM-integrin pairs leads to tight adherence by activated leucocytes and allows subsequent migration.

Endothelial Dysfunction

Endothelial dysfunction was initially recognised to occur in patients with established atherosclerosis. Elegant cardiac angiographic studies showed paradoxical

TABLE 1

CELL ADHESION MOLECULES

Major CAM Families

Selectins			
	L-selectin	P-selectin	E- selectin
Integrins			
	b1	VLA-4	
	b2	CD 11a/18	11b/18 11c/18
	b3	IIb/IIIa	Vibronectin receptor
Immunoglobulin superfamily			
	ICAM-1,2	VCAM	PECAM-1

Leukocyte Migration and CAM Pairs

Rolling		
L selectin	-	P & E-selectin
Triggering		
CD31	-	chemokines, integrins
Adhesion		
CD 11b/18	-	ICAM
VLA-4		VCAM
Migration		
CD31	-	extracellular mucopolysaccharides

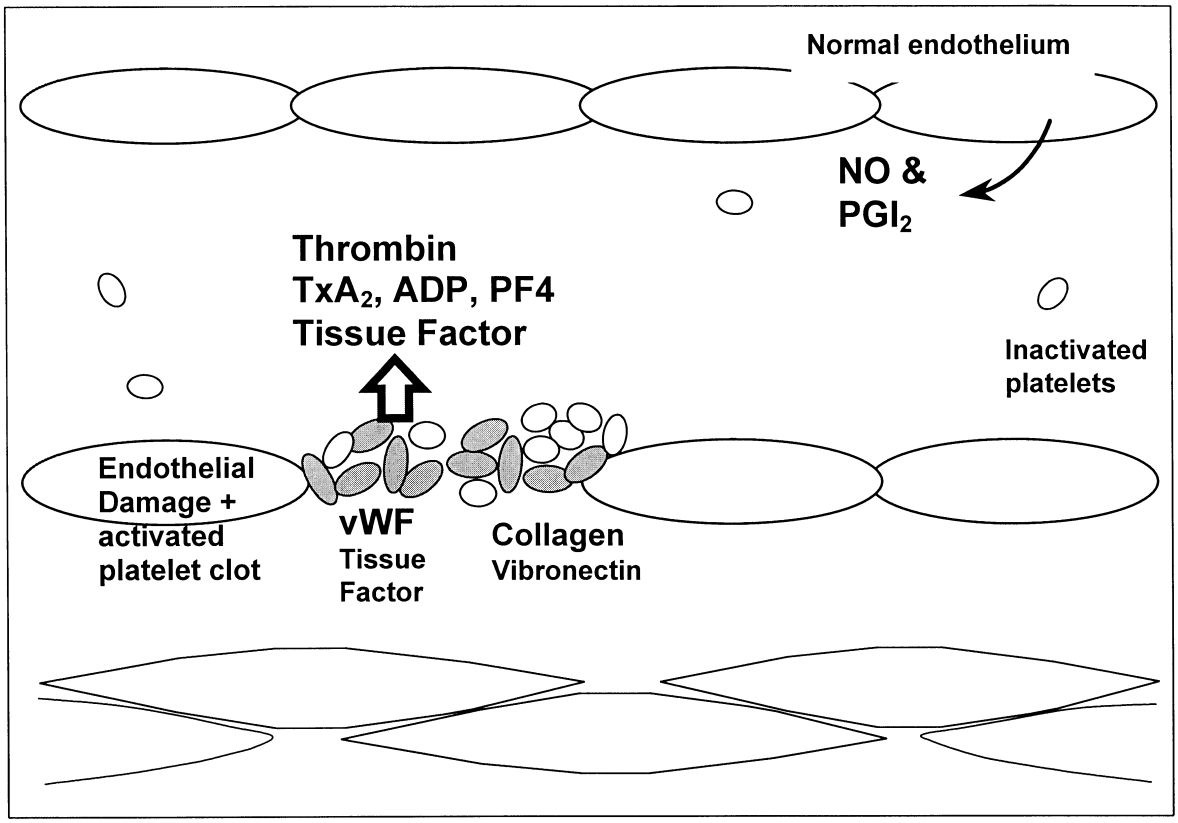

Figure 2. Formation of a platelet clot in response to endothelial injury.

vasoconstrictive responses to acetylcholine and serotonin. This was followed by the demonstration of exercise induced vasospasm, a contrast to the normal flow mediated vasodilatory response. It is now recognised that these phenomena are largely a reflection of impaired NO (and/or PGI_2) production by the endothelium at sites of atherosclerosis. Endothelial dysfunction can be measured by a number of non-invasive means, particularly by the use of flow mediated vasodilatation. These functional changes are seen in a number of conditions traditionally associated with a risk of subsequent vascular disease (Table 2). Endothelial dysfunction is now thought to be a marker for the development of atherosclerosis.

Impaired NO production is also associated with an increase in endothelial CAM production and hence leucocyte adhesiveness. Increasingly, inflammatory cells are recognised as playing an important role both in the development of atherosclerosis and in the conversion of stable atheroma into unstable disease. The changes in CAM production and in NO/PGI_2 release favour leucocyte accumulation. Lymphocyte actions within atheromatous plaque drive pro-coagulant pathways, such as macrophage TF production. When stimulated (e.g. by intercurrent infections) they release IFNg that is a major contributor to weakening of the plaque structure and increasing its propensity to plaque rupture.

Whether such endothelial dysfunction and/or atherosclerosis is a predisposition to other vascular disease states such as decompression illness can only be conjectured.

Reperfusion injury and cardiac bypass

Cardiopulmonary bypass (CPB) has long been recognised as causing a variety of acute vascular and haematological effects. These can be broadly separated into the effects of exposure of blood elements to the CPB circuitry and the effects of ischaemia-reperfusion in the coronary and pulmonary vascular beds.

Platelets bind to the synthetic materials of the by-pass circuit and this contributes to the mild fall in platelet count seen after CPB. Complement activation, with the generation of complement, leads to leucocyte activation, with subsequent generation of leukotriene B4 and increase in CD 11b/18 production.

During CPB, the ischaemic myocardial and pulmonary circulations increase P-selectin and ICAM production. Reperfusion of areas is associated with neutrophil sequestration and generation of activation products such as ROIs. In the lungs, increased endothelial permeability leads to the development of interstitial oedema and alveolar collapse, hypoxia and, in extreme cases, adult respiratory distress syndrome (ARDS). Similar reperfusion injury to the myocardium is associated with (transient) impairment of myocardial contraction (known to cardiologists as “stunning”).

There is increasing interest in the role of platelet-leucocyte interactions. Stimulated platelets can bind to neutrophils and cause their activation. In addition,

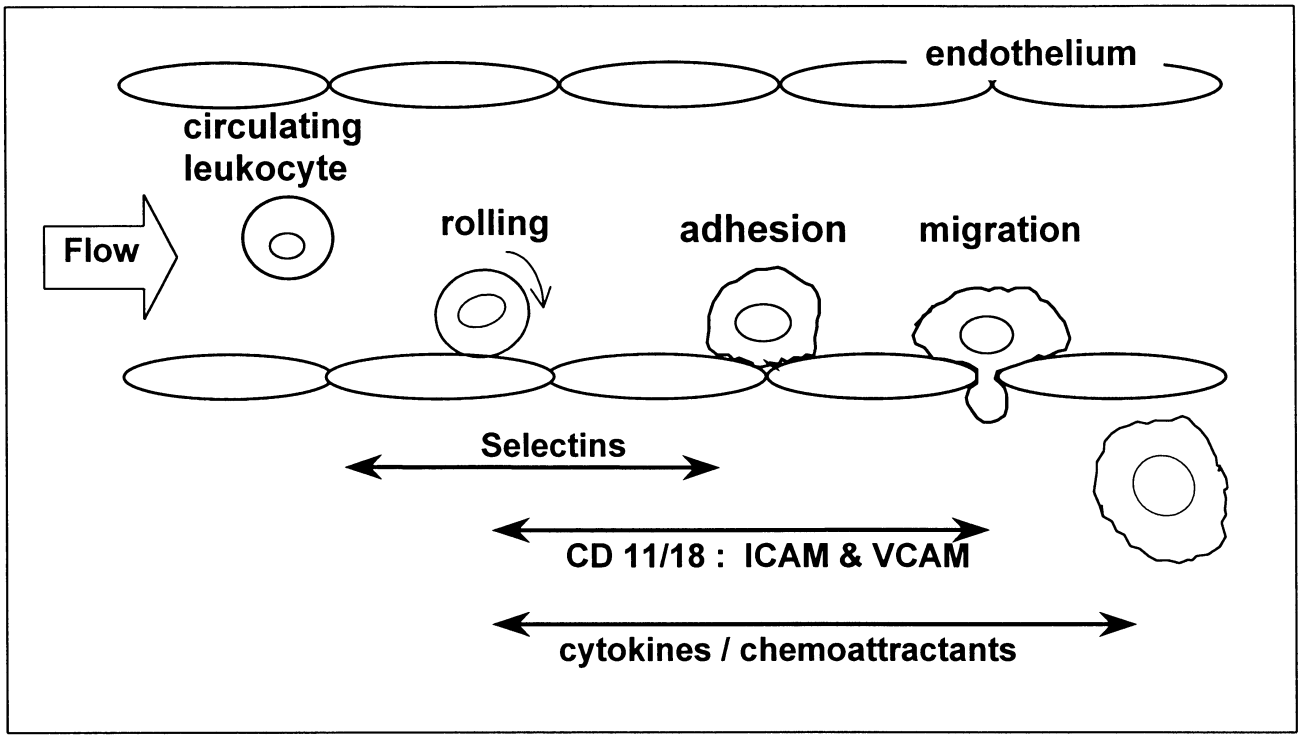

Figure 3. Schematic diagram of a leukocytes rolling on the endothelium, becoming activated by local cytokines and adhering to the endothelium, and then transmigrating out of the vascular space.

heparin, routinely given with CPB, causes platelet micro-aggregation. This effect is (partially) reversible by later protamine administration. Platelet micro-aggregates are readily demonstrable during CPB; they have the potential to cause microvascular obstruction and contribute to post-operative thrombocytopenia.

Doppler ultrasound has been used to identify and quantify vascular bubbles. The Doppler signal is thought to represent micro-bubbles from intravenous fluids, and during CPB, from "leak" of free gas from oxygenators. Both new cognitive neuropsychiatric deficits and MRI lesions after surgery correlate with the extent of Doppler signal intensity pre-CPB, raising the possibility that Doppler may also be detecting non-gaseous embolic material. At present there is no clear link between this observation and the phenomena of heparin-induced platelet aggregation or leucocyte/platelet interactions or activation.

Therapeutically, the administration of monoclonal antibodies against CD 11b/18 leads to a reduction in both neutrophil sequestration and hypoxia in animal models of CPB. In human observations, administration of an antibody to platelet gp IIb/IIIa (vide infra) has been associated with a reduction in post-CPB thrombocytopenia.

Decompression Illness

The nature of decompression illness has been partially elucidated in recent years. Our current understanding involves a bubble mediated endothelial injury that leads to neutrophil activation and adherence. The neutrophil response presumably occurs secondary to increased CAM and/or chemotactic cytokine production. Intra-cerebral vasospasm leads to the clinical syndrome of cerebral decompression sickness. The mechanism of neutrophil-induced vasospasm remains speculative. Neutrophil activation products such as ROIs are known to both inactivate NO and to inhibit NO synthesis by NOS. Additional vasospastic mechanisms are likely to be involved.

It is uncertain why some subjects form circulating bubbles after hyperbaric air exposure, and why despite bubbling being relatively common, only a small proportion of patients apparently suffer from these vasospastic phenomena. Also, the mechanism of benefit of hyperbaric oxygen therapy (HBO) remains speculative. Animal models of pulmonary bubble injury (air embolism) are associated with increased vascular permeability and this is exacerbated by neutrophil and complement activation. HBO can reduce neutrophil sequestration and activation in the rat gracilis muscle flap model of ischaemic-reperfusion, and can reduce b_2 integrin (CD 18) dependant binding of human neutrophils by a mechanism dependant on impairment of cGMP synthesis.

The potential benefits of HBO may be offset by the possibility of oxygen toxicity. HBO causes an oxidant stress to the lungs with increases in pulmonary neutrophil activation and ROI generation and with reduction of ROI catabolism (by phospholipase A_2). The neurological toxicity of oxygen is paradoxically associated with increased cerebral NO, and can be prevented by inhibitors of neuronal NOS or monoamine oxidase.

The mechanism of benefit with HBO may depend on the specific clinical circumstance in which it is being applied. There is increasing interest in the role of neutrophil antagonists to both reduce the vasospasm of DCI and prevent the toxicity of HBO.

New therapies

A variety of new therapies targeting adhesion molecules are currently being evaluated or entering clinical practice.

The first therapeutically useful anti-platelet agents are blockers of the gp IIb/IIIa receptor, which inhibit the final common pathway of platelet activation. ReoProÆ (generic name abciximab) is a chimeric antibody fragment; specific mouse anti-IIb/IIIa binding domains have been cloned onto human immunoglobulin constant regions to produce a minimally antigenic molecule. ReoProÆ given intravenously is a rapid acting, potent platelet antagonist that has proved useful in the treatment of complicated angioplasties. The next generation of specific, parenteral, non-protein IIb/IIIa blockers (e.g. tirofiban Æ produced by MSD) have completed phase 3 trials and may be available to the marketplace shortly, to be followed by orally active agents in the next 2-3 years.

Monoclonal antibodies have been shown to be useful in a variety of animal models of ischaemia-reperfusion. Similarly, soluble CAM fragments may prove useful as competitive antagonists of their counter receptors.

TABLE 2

ENDOTHELIAL DYSFUNCTION

Causes and Associations

Hypercholesterolaemia	Homocystine
Hyperglycaemia	Hyperinsulinaemia
Smoking	Oestrogen withdrawal ?
	Atheroma

Consequences

Reduced endothelial NO / PGI_2 production
paradoxical vasoconstrictive responses
Increased adhesion molecule expression
increased platelet & leukocyte adhesiveness
Susceptibility to vascular disease(s)

Soluble P-selectin has been used experimentally to bind to leucocyte L-selectin, thus preventing normal leucocyte rolling on activated endothelium. Antagonists of CAMs are likely to undergo a similar process of development as that of the IIb/IIIa blocking anti-platelet drugs.

Conclusions

The complex and interrelated functions of the endothelium in health are being slowly unravelled. With this our understanding of endothelial abnormalities in a variety of diverse disease states is increasing. In the near future we are likely to see major therapeutic advances based around the roles of these endothelial derived mediators.

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

OCCUPATIONAL DIVERS' KNOWLEDGE

Professional diver knowledge of diving medical issues, a pilot study.

Strauss MB, Borer RC Jr and Borer KM. *Undersea Hyperbaric Med* 1998; 25 (Suppl): 41

Abstract

Background

Professional divers are those individuals who receive monetary compensation for their diving activities. They are often unaware of the medical problems that may occur with diving. This pilot study examined professional diver knowledge of diving medical issues (DMI) through a questionnaire.

Methods

Twenty-six professional divers completed a 5-part diving experience questionnaire including ten statements on DMI. Four of the DMI statements reflected knowledge of general diving medical information (Group 1), three statements required specific diving medical knowledge (Group 2) and three statements dealt with unresolved diving medical controversies (Group 3). A single response from the following three choices: "agree" (A), "disagree" (D) and "not sure" (NS) was made for each statement.

Results

Professional divers showed a good knowledge of general medical issues in Group 1 responses: 88% correct, 5% incorrect and 7% NS. Controversial Group 3 statements had responses equally divided between the three choices: A=30 (39%), D:20 (26%) and NS:27 (35%). Responses to Group 2 statements regarding specific DMI were: 23% correct, 41% incorrect and 36% NS.

Conclusions

This pilot study reveals a lack of specific diving medical knowledge among professional divers. We feel this study should be expanded with a questionnaire given to each diver at the time of his or her interval diving physical examination. The diver's responses to the questionnaire should be discussed with him or her in order to make the diver as knowledgeable about medical aspects of professional diving activities as possible.

From

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Key Words

Occupational diving, underwater medicine.

CARBON MONOXIDE POISONING

Carbon monoxide poisoning in recreational diving: an uncommon but potentially fatal problem.

Caruso JL, Hobgood JA, Uguccioni DM and Dovenbarger JA. *Undersea Hyperbaric Med* 1998; 25 (Suppl): 52

Abstract

Background

Carbon monoxide (CO) is a colourless, tasteless, non-irritating gas that is produced by the incomplete combustion of hydrocarbons. It is the most common cause of death by poisoning in the United States. Extremely low levels of carbon monoxide arc normally present in the atmosphere, but great care is usually taken to avoid abnormally high amounts being introduced into scuba tanks. Increased lev-

els of carbon monoxide in the breathing gas of a diver can result in a potentially catastrophic outcome.

Methods

The Divers Alert Network (DAN) attempts to collect all available information on recreational diving fatalities that occur in the United States or those that involve US citizens diving abroad. When possible, DAN acquires the autopsy report and toxicology studies related to the fatality. A total of 451 fatalities in the DAN data base, occurring during the years 19931997, were reviewed to search for potential deaths related to carbon monoxide poisoning. Particular attention was paid to dive profile, the circumstances surrounding the fatality, autopsy findings, and post mortem carboxyhaemoglobin (COHb) levels.

Results

Of the 451 diving fatalities, COHb levels were known to have been measured in 67 (15%) cases. In 33 of these fatalities, the COHb levels were reported to be negative. In all of the remaining cases except two, the reported COHb levels ranged from 1-10%. COHb levels of less than 10% are not considered significant and may be found in asymptomatic individuals. There was a 1994 fatality for which the COHb level was found to be 32% at autopsy. Since CO poisoning was not suspected at the time of the mishap, the gas remaining in the tank was not analysed. In a fatality that occurred in 1997, the COHb level was also 32% and an evaluation of the decedent's tank, as well as the tanks of others in that dive group, showed elevated levels of carbon monoxide. An additional fatality, not included in this review, occurred in 1990 and involved a diver who mixed his own enriched air. The postmortem COHb level in that case was reported as 18%.

Conclusions

Despite the fact that CO poisoning is the most common cause of death due to poisoning, diving fatalities attributed to the effects of carbon monoxide are fortunately rare. Postmortem toxicology to measure the COHb level, however, is seldom performed in fatal diving mishaps. It is possible that some diving fatalities associated with CO toxicity are being missed. With an increased number of divers mixing their own breathing gas, carbon monoxide poisoning may become a more significant problem. Routine toxicology, including a COHb level, should be performed for most diving fatalities. This is especially true when the mishap is unwitnessed, or when there is any possibility that toxic substances may have impaired the diver's performance in the water.

From

The Divers Alert Network, Department of Pathology, and the FG Hall Hyperbaric Center, Duke University Medical Center, Durham, North Carolina 27710, USA.

Key Words

Carbon monoxide, deaths, recreational diving.

Autonomic neurotoxicity of jellyfish and marine animal venoms

Burnett JW, Weinrich D, Williamson JA, Fenner PJ, Lutz LL and Bloom DA.

Clin Auton Res 1998; 8: 125-130

Abstract

Venoms and poisons of jellyfish and other marine animals can induce damage to the human nervous and circulatory systems. Clues to the pathogenesis and clinical manifestations of these lesions can be obtained from data of human envenomations and animal experimentation. Because many investigators are unaware that marine animal venoms have autonomic actions, this paper aims to elucidate the broad antagonistic or toxic effects these compounds have on the autonomic nervous system. Marine venoms can affect ion transport of, particularly, sodium and calcium, induce channels or pores in neural and muscular cellular membranes, alter intracellular membranes of organelles and release mediators of inflammation. The box jellyfish, particularly *Chironex fleckeri*, in the Indo-Pacific region, is the world's most venomous marine animal and is responsible for autonomic disorders in patients. The symptoms induced by these venoms are vasospasm, cardiac irregularities, peripheral neuropathy, aphonia, ophthalmic abnormalities and parasympathetic dysautonomia. Cases of Irukandji syndrome, caused by the jellyfish *Carukia barnesi*, have symptoms that mimic excessive catecholamine release. Coelenterate venoms can also target the myocardium, Purkinje fibres, A-V node or aortic ring. Actions on nerves, as well as skeletal, smooth or cardiac muscle occur. Recent studies indicate that the hepatic P-450 enzyme family may be injured by these compounds. The multiplicity of these venom activities means that thorough understanding of the sting pathogenesis will be essential in devising effective therapies.

Key Words

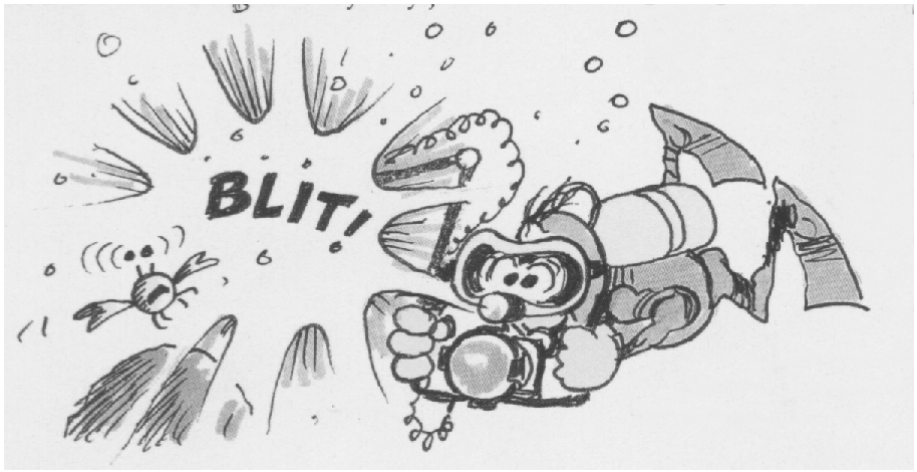
Marine animals, venoms, toxins, envenomation.

THE SEA PEOPLE'S GUIDE TO DIVERS
PART THREE

By RICO

Humans say that to see themselves as others see them is a great blessing. Imagine then what a blessing it would be to see themselves as other species see them. If only we could find a way of giving them a Sea People's view of themselves. Well, actually, we can...

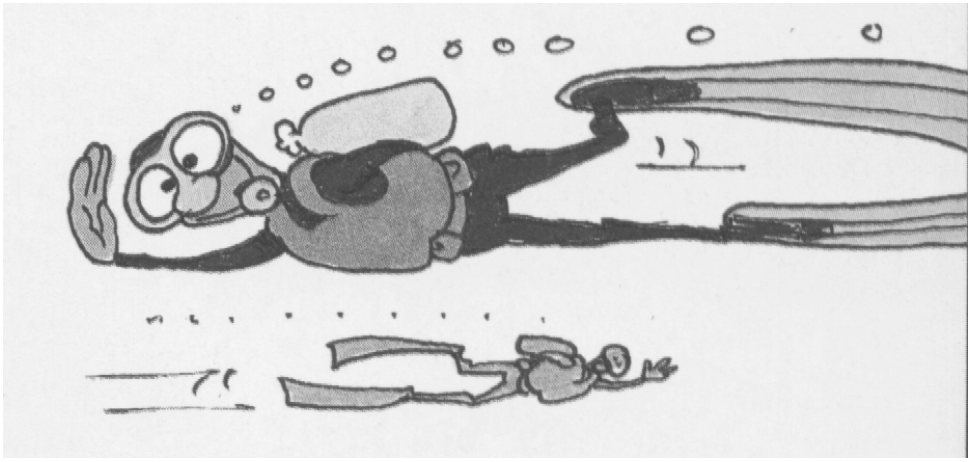
Thanks to the kindness of Rico, the cartoonist, and of Bernard Eaton, the Editor of DIVER, who have agreed to allow this series of typical divers to be reproduced in the SPUMS Journal. Although the featured diver types originated in the UK, we believe that most of them, at one time or another, have attended a SPUMS Annual Scientific Conference.



Flashgun Sniper

The Flashgun Sniper has ruined many an innocent invertebrate's day by cindering its tiny corneas with a million-candlepower light blast without warning. They risk frustration and hardship for their passion, and some have flooded their Nikonos so often that their cameras support inter-tidal communities. Their cameras and fancy equipment leave them scant time to consider the moralities of marine life, so copulate at your peril while they are about. Be alert for them! They may just pick you up and drop you in the middle of a colourful but deadly stinging anemone, for the sake of the "rule of thirds". If you are a sea urchin, for the sake of their art they might beat your head in with a rock, then decorate it with your major organs just to attract the pretty fishes.

Flying Darters



Flying Darters often accompany shoaling divers. Like the penguin's wings, their arms have lost most of their use and are generally held behind or clasped beneath them, while their extra-long foot-flukes propel them through the water. Curiosity is their driving instinct, so these flitting voyeurs get their highs from watching rather than doing. Sadly, their tiny attention spans allow them only brief surveillance at any point of interest, leaving them with no lasting information. Without this intellectual burden, they are left free to perfect the art of inane grinning and repetitive saluting typical of their kind.

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DIVER is published by Eaton Publications, 55 High Street, Teddington, Middlesex TW11 8HA, United Kingdom. The annual subscription is £ 33.00 which must be paid in English pounds.