The Editor's offering

A Happy Christmas and a Prosperous New Year to all our readers. It is the Editor's hope that this Journal will provide some interesting reading over the holiday period. Once again the Editor gives thanks to Drs John Couper-Smartt and David Davies for their invaluable assistance with the proof reading. Thanks are also due to those who have acted a peer reviewers. Due to the small size of the Australasian Diving and Hyperbaric World their names are not revealed, nor are those of the authors of the reviewed papers.

In 1996 eleven scuba divers and eleven snorkel users died in Australian waters. Never before have so many snorkel users died in one year. The reader may wonder why the term "snorkel users" is used. Only two of these people were actually breath-hold diving, the other 9 were tourists on the surface looking down. At least three of them were in water so shallow that their heads would have been out of the water if they had stood up. Snorkels must now be considered as dangerous equipment for those who have never used them before. Snorkel ends are not always above water and breathing in a mixture of air and water is an alarming incident if it is completely unexpected. The coughing and spluttering tends to occupy the swimmer's full attention and can easily lead to panic, which can lead to drowning. For more information about these snorkel and diving deaths turn to Dr Douglas Walker's diving deaths report on pages 182-196. Dr Carl Edmonds follows it up with a review of snorkel diving, which goes into the physiology of breath-hold diving and its perils of anoxic unconsciousness on ascent or after hyperventilation before taking the plunge. This is a practice to be discouraged for it contributes one or two deaths to every year's crop of diving fatalities, always an experienced and competent breath-hold diver.

We have a report on the technical diving and rebreather forum held in April 1999 in Sydney. Dr Lynn Taylor is not a medico, but then nor are most divers. Her non-medical review of the meeting can be found on pages 203-205.

The US now has a sub-speciality qualification in Undersea and Hyperbaric Medicine, retitled from Undersea Medicine. It requires the candidates to be Board Certified in some speciality before they can sit the examination. Hyperbaric medicine has spread very widely in the USA and quality control of physicians and facilities is on the agenda there as it is in Australia. Part two of the new Australian Standard for non-diving work in compressed air is going to provide standards of training for hyperbaric chamber operators, the attendants who care for the patients in the chambers and the doctors who treat them. The Special Interest Group in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists is working towards standards of training and career structure for diving and hyperbaric physicians. There is so much overlap in the composition of the two committees, which have different target markets, that divergence of opinions is very unlikely.

Those who attended the Palau meeting in 1998 will remember Des Gorman's presentation on the protective effects on neurological function of lignocaine infusions during cardiac surgery. These patients had open heart surgery with plenty of Doppler detectable bubbles in their carotid arteries, arterial gas emboli. The work was published in *The Annals of Thoracic Surgery* and we are indebted to Dr Thomas B Ferguson, the Editor, for permission to reprint the paper on pages 212-221. If lignocaine can preserve neurological function in the face of iatrogenic air emboli it might be able to do the same for divers with multiple small air emboli. The difficulty is assessing its results. Will they be the same when the lignocaine is given after some hours rather than at the time of the insults? Time, well designed experiments and careful clinical studies will eventually give the answer.

We review three excellent books in this issue. One, *Venomous Creatures of Australia* by Struan and John Sutherland, should be on the bookshelf of every doctor and in the pocket of every bushwalker or sea side visitor. It should be available in every house with a garden. Another is *Sea Snakes* by Harold Heatwole and the third is *20,000 Jobs under the Sea*, a history of diving and underwater engineering written by Torrance R Parker, a former commercial diver, who when sent a SPUMS Journal with the Editor's request for a review copy not only provided the book by return of post but included an application form to join SPUMS.

We publish the first of the papers from the 1999 meeting in Layang Layang. Dr Alf Brubakk opens the batting with a discussion of the effects of bubbles on the body. Dr Akin Toklu from Istanbul and Dr Gustavo Ambriz from Thailand both report series of DCI cases. Both record long delays between the onset of symptoms and arriving at the hyperbaric facility. Scandinavians provided 40% of Dr Ambriz's workload! Dr Mike Bennett proposes a possible method of excluding decompression illness as a diagnosis by studying the gutter of the lower eyelid with a slit-lamp looking for bubbles.

Among the Gleanings from Medical Journals is an abstract on action of hyperbaric oxygen unknown to the Editor, an oxygen induced osmosis which acts as a pump to remove fluid from injured soft tissue. The author, Professor Brian Hills, was SPUMS Guest Speaker in 1983. He suggests that an osmotic oxygen pump offers a better explanation of effects of hyperbaric oxygen than circulatory oxygen levels.

ORIGINAL PAPERS

AUSTRALIAN DIVING–RELATED DEATHS IN 1996

Douglas Walker

Key Words
Accidents, deaths.

Summary

This review discusses 11 snorkel-using swimmers and 11 divers using scuba. Only one of the snorkel users was experienced (BH 96/4) and the true reason for his death is unknown. The remainder fall into the category of almost total inexperience, calm sea, separation from others and silent death. By a strange chance there were two with an epileptic history in this group, BH 96/3 and BH 96/7, the former having also a history of previous myocardial infarct and the epilepsy being incidental rather than causative. Those having a duty of care as responsible for the safety of a group of persons swimming, some of whom are using a snorkel with minimal, or no experience, have an extremely difficult task in attempting to identify the sub group of such swimmers who show no outward signs of being in distress before silently dying. The scuba divers show a wide range of factors, inexperience, water power, excessive depth (perhaps by error), tight wet suit neck, and air embolism type symptoms.

Four of the inexperienced scuba divers ran out of air but none of the experienced divers actually ran out of air, indeed four of the five in this group had fully adequate remaining air. In contrast to the snorkel user group, health was less of a factor, with angina as a possible factor in one and psychological factors involved in another.

Breath-hold divers and snorkel users

BH 96/1

An overseas family visiting a popular island hired some masks and snorkels. One showed three others how to use the equipment and then he left them for 10-15 minutes to snorkel, by himself, a short distance away.

When he returned two of them were in shallow water and pointed to where the third person could be seen floating about 7 m from the beach in only 1 m of water. She was fit and a good swimmer but, as the end of her snorkel was at the surface of the water, he waded out to her, though not alarmed at that stage. When he lifted her head, blood and water came from her nose and mouth. He quickly dragged her ashore but resuscitation was unavailing. When he had last seen her she had been snorkelling in a normal manner, about 5 m off the beach, and he had watched her for about 5 minutes.

The cause of death was drowning and there were no adverse health factors. It is most likely that she got water down her snorkel and, because this was the first time she had used a snorkel, she failed to respond by blowing it clear, or by tearing off her mask and raising her head, and forgot the water was so shallow she could simply stand up.

FIRST USE SNORKEL. GOOD SWIMMER. HEALTHY. SILENT DROWNING AT SURFACE IN CALM, SHALLOW WATER CLOSE TO OTHERS.

BH 96/2

This group of overseas visitors had an interpreter with them but it is probable that they failed to pay attention to the information given to the passengers during the trip out to the Barrier Reef pontoon from which they were to view the reef. As the victim was a poor swimmer he chose to accept and wear a life vest but did not add fins to the mask and snorkel he used. There were crew members watching over the designated swimming area but their task was made very difficult because there were about 50 people in the water at any one time, with a constant flow of persons entering and leaving the water. The victim, floating 15-20 m from the pontoon with the end of his snorkel above the surface, was thought to be rather too still so the lifeguard entered the water and to check. He turned the victim face up and saw that he was unconscious. The snorkel was still in his mouth. Resuscitation was commenced as soon as he was lifted onto the pontoon and an initial response was obtained but not maintained. Some difficulty was experienced when lifting him from the water because he was liberally coated in sun tan lotion.

The autopsy showed there was almost complete occlusion of the left circumflex coronary artery and 60% narrowing of the left anterior descending coronary artery. However there was no histological evidence of myocardial ischaemic changes. He had suffered a stroke 2 years before and was taking medication for hypertension and to lower his cholesterol. No details of his recent health are known beyond the statement that he had some residual deficit from his stroke. From the history it is believed his death was due to a cardiac cause.

SNORKELLING WEARING A LIFE JACKET. SILENT SURFACE DEATH. IN CROWD. FLOATED FACE DOWN. SNORKEL STILL IN MOUTH. HISTORY OF STROKE, HYPERTENSION AND HYPER-CHOLESTEROLAEMIA. CORONARY ARTERY DISEASE. PRESUMED CARDIAC DEATH.

BH 96/3

Two overseas visitors, a man and his wife, went on a day trip to the Barrier Reef. After she had snorkelled she returned to the pontoon and gave him the mask and snorkel. One of the crew advised him to reposition the mask strap, advice he did not appreciate. He was watched for 3-5 minutes in the water by the lookout. His wife also watched him for a time, then both were distracted. A short time later the lookout saw him drifting at the surface with his head dipping from time to time. As the victim did not react to the end of the snorkel becoming submerged the alarm was raised. When he was reached he was unconscious. He failed to show any response to resuscitation.

It was later established that he had a history of epilepsy, starting in 1976. His last fit was in 1991 when he was taken to a hospital where myocardial ischaemia and possible evidence of a past (silent) myocardial infarction was noted. More recently, he had suffered an episode of mild left ventricular failure due to mitral regurgitation, thought to be a consequence of the previous myocardial infarct. Autopsy revealed a scar in the inter-ventricular septum but only mild coronary atheroma with patent vessels. There was marked atheroma in the aorta, iliac and cerebral vessels and cardiomegaly was reported. The left pleural space was obliterated. Although clinically acceptable, the official cause of death, acute myocardial infarct in association with marked coronary artery disease, was not supported by the recorded findings. While it is probable that he suffered a fatal cardiac event, it is possible his epilepsy had recurred. He wore dentures (upper and lower) but these were removed before the resuscitation efforts and were not an adverse factor.

SNORKELLING IN GROUP. SILENT DEATH. HISTORY OF EPILEPSY AND PAST MYOCARDIAL INFARCT. CARDIAC DECOMPENSATION EPISODE. SCARRING LEFT PLEURAL SPACE. PROBABLY ACUTE CARDIAC DEATH.

BH 96/4

Despite being unwell for 7 months with Chronic Fatigue Syndrome symptoms which followed a probable infection from Ross River Fever, he had been sufficiently fit to spear fish with his buddy. Both of them were experienced and capable of diving to 24 m (80 ft). The sea conditions were described a being ideal for diving and while two friends remained in the boat, ready to respond to any requests for assistance, the victim and his buddy began spearfishing about 200 m from the boat. They kept apart from each other for reasons of courtesy and safety. Each had a float with a line to his spear gun and placed fish on a line when caught. They were about 10 m apart until the buddy returned to the boat to have some lunch. The victim was seen from time to time at the surface, his fins being visible as he commenced each dive.

They heard a boat approach and saw it circle the diver's float, then it came close to their boat. It was the victim's habit to talk to any boat which came near and they had not seen him at the surface for some time, so they quickly motored over to the float and pulled it up. It was heavy. This was because the victim's body was caught on the (discharged) spear gun. He was not entangled nor tied to it in any way. His mask was half full of water. Not unexpectedly, he failed to respond to resuscitation. It is assumed that he drowned after suffering a post-hyperventilation blackout.

EXPERIENCED SPEARFISHERMAN. DELAY BEFORE ABSENCE NOTED. HAD FIRED SPEARGUN. FOUND WHEN SPEARGUN PULLED UP. POST-HYPERVENTILATION BLACKOUT.

BH 96/5

The members of a trade mission were taken to see the Barrier Reef. The visibility was poor so the glass bottomed boat trip was cancelled and they were offered the chance to go snorkelling. It is not known whether the victim and others in the party were in the saloon when a short talk on snorkelling was given to the passengers during the trip out to the island, but it is believed that one of the four heard the talk and passed on some information to the other three later, while they were on the beach. There were notices about the availability of instruction but their boat schedule prevented them from taking up the offer.

The water was shallow off this beach. The victim soon became separated from the others, who found that the wind made their return to the beach difficult. Probably at least 30 minutes passed before they saw him floating 10-15 m from the beach in waist deep water. They pulled him ashore but he did not respond to resuscitation. Autopsy failed to reveal any medical condition which could be implicated. No information is available concerning his swimming ability nor whether he had ever snorkelled previously. It is likely he had never previously used a snorkel and failed to respond correctly when he got water down his snorkel.

SNORKELLING. PROBABLY FIRST TIME. SHALLOW WATER. SOME CURRENT. SEPARATION FROM OTHERS. AFTER 30 MINUTES SEEN FLOATING UNCONSCIOUS. SILENT DROWNING. SWIMMERS NEARBY UNAWARE OF TROUBLE.

BH 96/6

This day trip brought its passengers to a cay among the reefs. During the trip out a talk on the basic safety rules on the boat was given and passengers were asked to fill in a medical questionnaire if they intended to join one of the "scuba experience" (Resort) dives. Then a diving instructor gave a talk about snorkelling and scuba diving.

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and Experience		Dive	Dive	Depth in metres		Weights	
		Victim	Buddy	group	purpose	Water	Incident	On	kg
BH 96/1	72	No training No experience	No buddy	Solo	Recreation	1	Surface	None	None
BH 96/2	51	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 96/3	79	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 96/4	33	No training Experienced	No training Experienced	Buddy Separation before incident	Spear fishing	18	Not stated	On	Not stated
BH 96/5	42	No training No experience	No training No experience	Group Separation before incident	Recreation	1.5	Surface	None	None
BH 96/6	19	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 96/7	20	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 96/8	24	No training No experience	No training No experience	Buddy Separation before incident	Recreation	1	Surface	None	None
BH 96/9	57	No training No experience	No training No experience	Buddy Separation before incident	Recreation	Not stated	Surface	None	None
BH 96/10	25	Training and Experience not stated	Training and Experience not stated	Buddy Separation during incident	Recreation	2	Surface	None	None
BH 96/11	55	No training Experienced	No Buddy	Solo	Hunting octopus	Not stated	Surface	On	16
SC 96/1	41	Trained Some experience	Trained Experienced	Buddy Separation before incident	Recreation	8	Surface	On	Not stated
SC 96/2	37	Trained Experienced	Trained Experienced	Group Separation before incident	Recreation	33 (Planned 27 m)	4.5	Off	Not stated
SC 96/3	45	Trained Very experienced	Trained Very experienced	Group Separation before incident	Recreation	76	Surface	On	Not stated

DIVING-RELATED DEATHS IN 1996

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	Comments
None	Not applicable	Not applicable	Not applicable	Hired	1st use of snorkel. Good swimmer. Shallow calm water. Silent drowning.
None	Not applicable	Not applicable	Not applicable	Hired	Solo in group. Poor swimmer. Valiant rescue attempts. Cardiac type death. Previous CVA.
None	Not applicable	Not applicable	Not applicable	Borrowed	Silent death in crowd. Epileptic. Previous myocardial infarction. Cardiac death.
None	Not applicable	Not applicable	Not applicable	Hired	Solo. Post-hyperventilation blackout. Recent chronic fatigue syndrome.
None	Not applicable	Not applicable	Not applicable	Hired	1st use of snorkel. Group separation. Silent surface drowning. Shallow water.
None	Not applicable	Not applicable	Not applicable	Hired	Inexperienced. Separation from others. Calm sea. Found drowned.
None	Not applicable	Not applicable	Not applicable	Hired	Silent death in group after resort scuba dive. Undeclared epilepsy.
None	Not applicable	Not applicable	Not applicable	Hired	1st use of snorkel. Buddy close. Poor health? Fatigue. Shallow water. Silent death
None	Not applicable	Not applicable	Not applicable	Hired	2nd use of snorkel. Separation from group. Cause of death uncertain.
None	Not applicable	Not applicable	Not applicable	Hired	Buddy went ashore but other swimmers were nearby. Silent drowning at surface.
None	Not applicable	Not applicable	Not applicable	Owned	History of significant ill health. Cardiac death.
Not inflated	Yes	Adequate	Some adverse	Owned	No dives for 5 years. Tight hood. Mask filled with water. Died during surface swim back to boat.
Inflated	Yes	Low	Adequate	Owned	Psychological and psychiatric problems. Possible asthma history. Ill health? Separation, then solo. CAGE.
Not inflated	Yes	Not stated	Equipment lost	Owned	Heavy equipment. Rough surface. Tried to swim into current, sank. Tight neck seal.

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and Experience		Dive	Dive	Depth in metres		Weights	
		Victim	Buddy	group	purpose	Water	Incident	On	kg
SC 96/4	40	Trained Some experience	Trained Some experience	Buddy Separation before incident	Recreation	30	Ascent	On	6
SC 96/5	46	Trained Inexperienced	Trained Very experienced	Group No separation	Pupil Advanced Diver	30	30	On	Not stated
SC 96/6	36	No traininng Experienced	No buddy	Solo	Cray fishing	3.5	3.5	On	15
SC 96/7	19	Trained Some experience	Trained Very experienced	Group Separation before incident	Work	9	Ascent	On	10
SC 96/8	38	Trained Experienced	Trained Experienced	Buddy Separation before incident	Recreation	10	Surface	On	Not stated
SC 96/9	32	Trained Experienced	Trained Experienced	Group Separation before incident	Recreation	32	22	On	8
SC 96/10	42	Trained Some experience	Trained Some experience	Buddy Separation during incident	Recreation	32	15	On	12
Sc 96/11	28	No traininng Some hookah experience	Trained Experienced	Buddy Separation during incident	Tuna farming in cage	20	20	On	7

Those who signed up for the scuba dive were ferried to the cay with the equipment. Those who were intending only to snorkel swam to the beach, a distance of about 150 m. There was at least one other boat with its passengers on the cay at this time. The victim told her friend she had used a snorkel previously. Her friend was making her first snorkel swim and experienced difficulties, so chose to wait to be taken ashore in the ship's dinghy when it had serviced the scuba diving passengers. She last saw the victim when the victim had swum about half way to the beach. Although they had signed up for the scuba they were not in the first group so were to snorkel first. The buddy joined those listening to the instructor, on the beach, about use of scuba and expected to be joined there by her friend. It was here that she was told her friend had been found floating, unconscious and had died.

The skipper of the other boat saw a person floating at the surface. He became alarmed when he saw there was no response when his boat's wake passed over the snorkeller, so he took his dinghy and investigated. She was unconscious and he bumped her head while pulling her limp body into the dinghy. CPR was unsuccessful. The pathologist maintained that the head injury occurred before her death, the skipper equally certain that his boat never hit her and that the injury occurred during retrieval. The skipper's version was accepted, but the pathologist described the blow as having been forceful and, he thought, significant. She was using her own mask, snorkel and fins but was wearing a wet suit provided by the boat. Her experience using a snorkel is not known but possibly was not great. She was described as being "an average swimmer". There was no adequate safety watch on those

DIVING-RELATED DEATHS IN 1996 (Continued)

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	Comments
Not inflated	Yes	None	Adequate	Owned	12th dive. Dived with buddy without instructor. Low air ascent. Separation. CAGE.
Not inflated	Yes	Adequate	Adequate	Dive shop	5th dive, on advanced course. Panic ? From cardiac pain? CAGE.
Not inflated	Yes	Adequate	Adequate	Owned	Solo. Cray fishing. Experienced. Tired. Possibly lost regulator from mouth.
Inflation failed	Yes	None	Some adverse comments	Work	Altered dive log to falsify experience. Out of air panic ascent. CAGE. History of cold water asthma.
Part inflated	Yes	Adequate	Adequate	Owned	Wrong underwater direction led to surfacing in surf zone. No dives in previous 12 months.
Not inflated	Yes	Adequate	Adequate	Owned	Trio separated during descent and failed to reascend together. Shot line incorrectly placed. Deep so nitrogen narcosis. Tight wet suit? Tired by surface swim.
Not inflated	Yes	None	Adequate	Hired	'Advanced diver' after 9 dives in 2 months. Over confident. Out of air. Deep dive. failed buddy breath as different BCD. CAGE death as boarding boat. Twice ran out of air during training.
Not inflated	Yes	None	Some adverse	Employer	Working inside 20 m deep fish pen. 1st scuba dive . Untrained. Some hookah experience. Strong current, poor visibility. Heavy air use. Out of air ascent. CAGE.

swimming to the beach from the boat. The cause of death was drowning, the only identified reason for this being the (possible) head injury.

SEPARATED. PROBABLY INEXPERIENCED WITH SNORKEL. NEAR OTHERS. SCALP INJURY OF UNCERTAIN SIGNIFICANCE. DROWNED.

BH 96/7

The two overseas visitors had met recently and, being from the same country, had decided to take a trip to see the Barrier Reef together. One of them had a history of frequent epileptic fits, as her companion knew. Indeed she had a fit the evening before they made the fatal trip. The passengers received the regular talk on shipboard safety. On the outward trip a medical history form was given to those showing an interest in taking the opportunity to make a supervised scuba dive. The victim asked her companion whether she should write down about her epilepsy, a question which he said was for her to decide. There is a difference of opinion as to whether she discussed the matter with the instructor. Her experience during her first "resort dive" was such that she signed up for a second one, the same group of passengers undertaking both trouble free dives. Strictly speaking the instructor had too many in his group, because his assistants lacked the necessary training and certification. His instructor organisation would not have supported his actions had any incident occurred. The victim's group was given an introductory talk on scuba diving while those who had chosen to limit themselves to snorkelling were given a talk on this subject.

When they returned from the second scuba dive they were told they could, if they hurried, join the snorkel group. This was led by the skipper. They were taken in the ship's dinghy to join the snorkel group off the beach. This escorted tour of snorkel using swimmers ended about 100 m from the boat and they were left to make their own way back from there. There was no crew member tasked with the sole duty of watching over the swimmers and it was only after a roll call was made of passengers and a search of the boat failed to find her that her absence was noted. One of the crew climbed the mast and he saw her body floating at the surface. Examination of her snorkel showed that one lug of her mouthpiece had been bitten off (it was in her mouth) so it was believed that she had suffered an epileptic fit and drowned. The diving instructor was noted to be using unpaid divers as assistants on the understanding that their work time would contribute to qualifying towards qualification to become dive masters, but he was not covered for this by the organisation he claimed would give the desired certification, though an "active" instructor in another.

The details of this case are incomplete as the skipper and the instructor claimed "privilege" and elected not to give any statements. The skipper had led the snorkel group on its tour to view the corals but his official terms of employment prohibited him from leaving the boat. Although those who chose to snorkel and not scuba were given a talk, the latter group received no instruction. It is not known whether the victim had any previous experience in the use of a snorkel but she apparently experienced no problems with its use. Her lack of concern about the implications to her safety of fits in the water is very difficult to understand but possibly reflected a desire to ignore (as far as possible) the reality of her medical problem. Her companion stated that she had a short warning of the onset of her fits and may have relied on this if she gave any thought to the possible risks. Being alone when the fit occurred removed the last possible safety factor. The passengers were not, apparently, advised to swim with another person as a buddy.

EPILEPTIC. FAILED TO DECLARE HEALTH RECORD. RECENT FITS. 2 RESORT DIVES, THEN JOINED SNORKEL GROUP. SEPARATION. SILENT, SURFACE DROWNING. INADEQUATE SAFETY WATCH OF PASSENGERS. INSTRUCTOR USED UNQUALIFIED ASSISTANTS.

BH 96/8

Here again two visitors from the same overseas country met by chance and decided to join one of the day trips to one of the Barrier Reef resort islands in order to go snorkelling there. They hired masks, snorkels and fins once they arrived and were given some instructions by the attendant in the shop. Because the victim understood English better than her companion she translated this advice to her, thereby giving her the impression she was knowledgeable about snorkelling. There was a long walk from the shop to the chosen beach and the victim was noted to become easily tired, though she did not mention having any ill health. When they came to don the equipment it became obvious that the victim was ignorant of its use.

After they entered the water they remained in the shallows, though moving away from others who were in the water because the victim found herself bumping into them. About half an hour later others saw the victim's companion walking along the beach and looking out to sea as if trying to find someone, then heard her scream for assistance. The victim was then seen floating face down in shallow water near some rocks. The water had been calm, the wash from boats entering the bay had not been troublesome and none of the boats came near the swimmers at the beach. It is not known how or why the two became separated but it is apparent the victim drowned silently at the surface and was probably fairly close to her buddy. Although there were guided snorkel tours they had just missed one and the time for return to the boat made the next tour too late. The autopsy revealed no reason for her becoming tired too easily and it is assumed that she drowned following inhalation of water down her snorkel, despite it having a purge valve.

FIRST EXPERIENCE WITH SNORKEL. SILENT, SURFACE DROWNING IN CALM SHALLOW WATER. MAY HAVE BEEN UNFIT. NO DISEASE FOUND.

BH 96/9

This group from overseas joined a trip to the Barrier Reef in the afternoon, after a morning spent "white water" rafting. At the island, they hired snorkelling equipment and swam off a beach with many other day trip visitors. There was a marker buoy where the water was deeper but most visitors were in waist deep water near to the beach. The victim was initially with his wife and daughter but after a time they returned to the beach to sun bathe. His wife became alarmed when he had not joined her after 20 minutes and she was unable to identify him among the swimmers. The two women started to walk along the beach to look for him and soon came across a group trying to resuscitate someone on the beach. He had been seen, by a tender taking passengers back to another boat, floating motionless, face down, some 500 m off the beach. It is probable he was dead when located. Getting him aboard the tender was difficult because of the sun tan oil on his body. No ill health was found at the autopsy and he had a history of regular health checks. It is possible that his inexperience with use of a snorkel was the reason he drowned as it was only the second time he had used a snorkel. Although a talk on snorkel use was given in his language during the boat trip, and similar instruction given when he hired the equipment on the island, his wife later claimed that no information was given. This illustrated the difficulty of ensuring that information is imparted effectively.

While there was a confirmation by the histologist that the victim had suffered a minor subarachnoid haemorrhage (without brain damage), and the pathologist firmly believed there had been a significant, but unrecorded, head injury at some time prior to death, there is no convincing evidence to show that this was of significance. The coronary arteries were healthy and no myocardial ischaemic changes were found. There was a large warty polypoid vegetation on the anterior pulmonary valve leaflet, thought to have led to the thickening of the left ventricle's wall. It was not thought to be a factor in this death. It is likely that his lack of familiarity with the use of a snorkel was the critical factor. When the head injury occurred was never decided.

SECOND USE OF SNORKEL. SEPARATION FROM FAMILY. IN A CROWD. IN CALM SHALLOW WATER. SILENT DROWNING. FOUND BY CHANCE. FLOATING FACE DOWN. NON-CRITICAL LESION OF PULMONARY VALVE.

BH 96/10

Shortly after she arrived from overseas this visitor met a compatriot and they decided to visit a Barrier Reef island together. On their arrival they hired masks, snorkels, fins and buoyancy vests. However she found her fins were too tight and soon discarded them. There were other swimmers snorkelling off this beach so her new companion had no fears about leaving her to lie on the beach and sunbathe. It was some time later that he became aware that he could not see her among the swimmers and he gradually became worried by her absence. One of the island's staff was sitting on the beach at this time and he noticed a person floating quietly among the swimmers, not reacting when others passed close by. He thought this was strange and decided to check, so swam out to her. He found her floating face down, snorkel out of her mouth and its end underwater. After turning her face up he towed her to the shore and commenced CPR, but there was no response. The buoyancy vest had unfortunately kept her floating face down. No health factor was found at the autopsy and it must be assumed that she had drowned after aspirating water down the snorkel. The water was only waist deep where they had been initially but where she was found it was 2.1 m (7 ft) deep so she could not have stood up when in trouble. Nothing is known about either her swimming ability or whether she had ever previously used a snorkel but gross inexperience seems to have been the critical factor.

SNORKEL EXPERIENCE NOT STATED, BUT POSSIBLY FIRST USE. SEPARATED FROM COMPANION. SILENT, SURFACE DROWNING IN CROWD OF SWIMMERS. NO FINS. VEST FLOATED HER FACE DOWN.

BH 96/11

It was customary in his native country to dive for octopus for food and he had continued this despite his probable awareness that it was not permitted in Australia in the areas where he liked to hunt. His wife had tried to dissuade him from diving alone and he had responded, as on this occasion, by hiding his intentions from her. His failure to return home at the expected time led members of the family to institute a private search of the locations he chiefly favoured but they drew a blank. By the time they notified his absence to the police the latter were trying to identify a body found in the tidal harbour that evening.

The finder was a man who had entered the water for a swim after spending some time relaxing on the beach. He had seen a float when he first arrived but taken no notice of it till he entered the water and then saw there was a body attached and it was slowly drifting with the incoming tide. He pulled it to the shore and hurriedly notified the police. They found that the victim must have entered the water some distance from where he was found, an indication that he was aware his activities were best kept unobserved. He was found to have a plastic bottle with him. This had possibly contained a solution to cause the octopuses to leave their crevices, and he had caught several before he died.

Autopsy showed that he had fibrosis of his lungs, 90% narrowing of the right coronary artery and up to 70% of the left, and the left ventricle showed thinning of the anterior wall and scarring of the lateral. His medical history was of silicosis, a myocardial infarct and prostatic cancer in remission. Despite his ill health history he had persisted to work long after others would have given up. Cause of death was given as atherosclerotic cardiovascular disease.

SOLO BREATH-HOLD DIVING (ILLEGALLY) FOR OCTOPUSES. SIGNIFICANT HISTORY OF ILLNESS. FOUND FLOATING. EVIDENCE OLD MYOCARDIAL DAMAGE. CORONARY ARTERY DISEASE. CARDIAC TYPE DEATH.

Scuba divers

SC 96/1

Although he trained in 1980 and dived frequently for about 8 years, he had rarely dived since his marriage in 1987 and had not dived at all for 5 to 6 years. However he had kept his tank "in test" and had replaced the O rings and obtained new straps for his fins before making this dive. His buddy had made 16 dives since qualifying 2 years before. The chosen dive site was an underwater track popular with local divers. Having gained weight, the victim had some difficulty getting into his wet suit jacket, but managed. The hood was tight, according to later witnesses. They entered the water from a ramp, descending to the chain at about 8 m. After about 5 minutes the victim

indicated that he wished to ascend, which they did without haste. He explained that his mask was filling with water and he did not wish to continue, so they started a surface swim back to shore. He started using his snorkel but soon changed over to scuba. When they were about 10 m from the shore, where the water was rougher, they lost contact with each other.

It was only after the buddy had come ashore, onto some rocks, and removed his equipment that he looked back and saw his friend floating at the surface, face up. A diving instructor, waiting for his class, also noticed the victim floating face up, moving passively with the surge. He quickly motored over to him, dived in and commenced in-water EAR. Others now came to assist and tow the victim to the rocks (his weight belt was probably ditched at this time). The buoyancy vest was noted to be in poor condition but this was not a factor as he floated face up.

The autopsy revealed neither the classical signs of drowning nor any significant coronary artery disease, although the heart was said to show mild cardiomegaly. This was possibly a cardiac death from arrhythmia in association with the effort of swimming in rough water, with possibly some aspiration of water.

SCUBA TRAINED. HAD NOT DIVED FOR 5-6 YEARS. SURFACE SEPARATION IN ROUGH WATER. BUOYANCY VEST IN POOR CONDITION. CAUSE OF DEATH UNCERTAIN. MILD CARDIOMEGALY. HEALTHY CORONARY ARTERIES. POSSIBLY CARDIAC FACTOR OR 'DRY' DROWNING.

SC 96/2

The basic facts are known, but the complete story is unlikely ever to be known because the victim was a very determined and intelligent woman who retailed a different medical history to different friends, whom she managed to keep apart. She claimed to suffer from multiple sclerosis and to have spent time in a wheel chair. She also claimed to have screws in her spine for vertebral changes and was thought to have suffered from depression and possibly asthma symptoms. She claimed to have received her basic training after obtaining a medical clearance, but there is no documentation to either confirm or deny any of these statements. Because she was (de facto) scuba diving, she was given further training to make her activities less dangerous to herself. This instructor was aware, in part, of her personality.

The fatal dive was to be to 27 m but was changed to a deeper one as the instructor had two pupils needing a "deep dive". She agreed to a 35 m dive on a reef. The descent down the anchor line was tiring as there was a strong current. She only descended 5 m before returning to the surface. Although the pupils stated that the instructor took her back to the surface he said he only became aware of her absence when they reached 20 m. He was not worried as there were two experienced crew in the dive boat who could take care of her.

The crew said that she seemed to be rather puffed before her initial descent but had refused their suggestion she wait in the boat. She dived again after a short rest and was not seen by the other three divers during their dive. She surfaced, while they were taking their decompression stop, about 50 m from the dive boat and appeared to be upright as she gave an "OK" signal. After picking up the three divers the boat moved to where she had been seen and found her floating there, dead. The autopsy confirmed that this was a cerebral arterial gas embolism death. Her booking for this dive was accepted without planning for her to be provided with a buddy. There was still 50 bar air remaining in her tank when it was checked.

TRAINING HISTORY IRREGULAR, BUT EXPERIENCED DIVER AND INTELLIGENT. HEALTH HISTORY SIGNIFICANT BUT UNDOCUMENTED. SEPARATION EARLY IN DESCENT. THEN SOLO DIVE. POSSIBLE HISTORY OF ASTHMA. BREATHLESS BEFORE DESCENT. POSSIBLE NEUROLOGICAL AND DEPRESSION HISTORY. X-RAY PROOF OF CAGE. PERSONALITY FACTORS SIGNIFICANT.

SC 96/3

All five of the divers making this deep dive were experienced in use of nitrox and trimix and they had all previously dived with each other. Since the recent death of a friend making a deep dive, the victim had been obsessional over safety, carefully planning his dives in advance and then calculating and making the appropriate gas mixture. Although there was a current they were confident that it would prove to be no problem. They anchored by snagging the anchor on the wreck, at 73 m, then let down two shot lines for their planned decompression stops. The longer one, from the stern of the boat, had a cross line to the anchor line so that after water entry at the stern they would be able to reach the anchor line more easily and so start their descent down it. A mermaid (safety) line was streamed from the stern.

The victim was the second to enter the water and he held onto the mermaid line, moving back along it to allow the third diver room to enter the water and to receive his video camera equipment. Meanwhile the first diver had reached the sea bed. A struggle with the equipment in the current caused the third diver to abort his dive at 54 m. These two divers could see each other but neither ever saw the victim after they left the surface. The anchor pulled free so divers one and three aborted the dive and decompressed on the shot lines. The crew saw the victim drift beyond the end of the mermaid line. He seemed to be attempting to orally inflate his buoyancy vest, then sank.

He was never seen again. His torn dry suit was found on the sea bed two days later but neither his equipment not any part of his body was ever found. It was accepted that the dry suit was savaged by sharks after his death. It is supposed that he may have failed to open the valve supplying air to his buoyancy vest but needed positive buoyancy. As he was negatively weighted, had the regulator out of his mouth while he was trying to inflate his vest, and had lost the "surface anchor" benefit of the mermaid line, he may have died because of a sudden submersion at this time.

EXPERIENCED DEEP DIVER. INTENDED DEEP DIVE WITH TRIMIX. SURFACE LOSS OF GRIP ON MERMAID LINE. NEGATIVELY BUOYANT. ATTEMPTED ORAL INFLATION OF BUOYANCY VEST. SEPARATION FROM BUDDIES AND BOAT. STRONG CURRENT. ANCHOR CAME FREE FROM WRECK. NEW DRY SUIT, POSSIBLY WITH TIGHT NECK SEAL. BODY NEVER RECOVERED.

SC 96/4

The two dive boats carried 15 divers in addition to an instructor and a dive master. The plan was for the more experienced to be guided through a passage in a large rock while the others were escorted to a more scenic, and shallower, adjacent area. The victim and her buddy were in a group of 4 or 5 at the surface with the instructor when one of the "passage" divers surfaced and asked the instructor to descend with him to assess whether he should attempt the deeper dive. The instructor told his group to wait for his return and descended with this diver. When he surfaced the victim and her buddy were no longer at the surface, having apparently decided they were competent to dive without supervision.

The victim had been diving for 3 months and had made possibly 11 scuba dives. Her buddy had been diving for 12 months but no details are available of her diving experience. They made an uneventful dive to 30 m, disregarding the 18 m depth limit of their certification level, and when the buddy noticed that her contents gauge showed 50 bar she indicated that they should ascend. She reported that the victim's gauge showed 150 bar, a degree of nitrogen narcosis probably influencing her acceptance of such a reading at this stage of their dive. First one, then the other, led their ascent, the buddy being in advance as they neared the surface. The victim failed to surface but there was no immediate alarm at this, it being assumed that she had boarded the other boat when she was not seen at the surface. After about 20 minutes delay the instructor decided to make an underwater search and found her lying on the sea bed, weight belt on and her tank empty (so he could not inflate her BCD).

Examination of her equipment showed it to function correctly after the tank was filled. An X-ray of the body taken before commencing the autopsy showed air in both ventricles and right atrium, the neck veins, the bile system and portal system. The autopsy showed also air in the Inferior Vena Cava, a small left sided pneumothorax, surgical emphysema in the neck, and a possible perforated left eardrum. It is assumed that she ran out of air and suffered a massive air embolism before reaching the surface. A friend stated that she had previously shown coolness in a stressful diving situation. The critical factors were failure to monitor her contents gauge, separation and possible nitrogen narcosis impairment in an inexperienced diver.

TRAINED. TWELFTH SCUBA DIVE. IGNORED INSTRUCTOR'S ADVICE TO DIVE WITH HIM. CERTIFIED TO 18 m. DIVED TO 30 m. SEPARATION DURING LOW AIR ASCENT. OUT OF AIR. MASSIVE AIR EMBOLISM. CAGE.

SC 96/5

The diving history of this unfortunate man lasted 8 days. He started an "advanced diver" course immediately he completed his basic course, during which he made four (4) dives. The instructor had four students and each made a giant stride water entry and waited on the mermaid line before they descended as a group. They stopped at 15 m to allow one of the group to equalise, then collected at the anchor while the instructor attached a come-back line to it as the visibility was poor. He then indicated they should follow him to an area 2-3 m away where the visibility was better. On looking back he saw that the victim had remained close to the anchor, though not holding onto it and was holding his regulator in his mouth with one hand. The instructor signed to the other three pupils to remain where they were and returned to the victim. He noticed he looked distressed and wide eyed, so decided he would take him to the surface. Having made the decision to abandon the dive, the instructor signed to the other three to follow him (they did not observe this signal) and started to ascend with the victim, who kept one hand on the line. As they ascended the instructor kept his legs round the line and arms around the victim. When they reached 10 m the victim removed his regulator but retained his grasp on it. He refused to allow it to be replaced in his mouth. At this time he seemed to be conscious. The instructor brought him up to the surface as quickly as possible and there ditched his weight belt while trying to keep his face above the surface. Now the victim was unconscious. In-water EAR was attempted before the victim was pulled into the boat and CPR commenced by the boatman, while the instructor descended to retrieve his three other pupils. Resuscitation efforts were unsuccessful.

Pre-autopsy X-ray films were taken and showed air in both ventricles and right auricle. The autopsy findings confirmed this and also found changes indicative of pulmonary barotrauma. The coronary arteries showed

areas of 70% narrowing and there were left ventricle wall changes probably indicative of a myocardial infarct. It is assumed that he had a pre-training medical check and neither revealed cardiac symptoms nor had any disease discovered. Possibly anxiety led to angina when he reached the sea bed, and his change in behaviour at 10 m was due to a myocardial infarct. The pulmonary barotrauma and arterial gas embolism were the consequence of his ascent the last 10 m while unconscious.

JUST TRAINED. NOW TAKING ADVANCED DIVER COURSE. INEXPERIENCED. MAKING FIFTH SCUBA DIVE. DISTRESS. POSSIBLE ANGINA PAIN. PANIC. CORRECT INSTRUCTOR RESPONSE. ASCENT WITH CLOSE CONTACT. VICTIM REMOVED REGULATOR AND REFUSED TO ALLOW REPLACEMENT. RAPID ASCENT LAST 10 m. EVIDENCE OF AIR EMBOLISM AND PULMONARY BAROTRAUMA WITH CAGE.

SC 96/6

This experienced diver had never received formal training but this did not prevent him obtaining air or buying new equipment. Indeed he intended to try out his new buoyancy vest on this dive and had added some 7 lbs to his weight belt for this reason. His wife helped him carry his diving equipment over the rocks to the water's edge, a walk which left him red faced and sweating. He admitted to a racing heart. Although he claimed to be feeling relaxed his wife was not convinced this was true. He indicated he would return from his solo dive in an hour so his wife sat on the rocks for about hour, then returned. She watched his snorkel for about 15 minutes before she realised that it was floating by itself, not attached to her husband. She raised the alarm and an air search was commenced which located his body in 3.6-4.5 m (12-15 ft) of water. His fins were missing, his legs were under a ledge, and there were crayfish spines in his (remaining) glove. His regulator was floating free so it was thought probable that it had come out of his mouth and he had failed to regain it in time to avoid drowning. He had 2/3 of his air remaining.

EXPERIENCED SCUBA DIVER. NO FORMAL TRAINING. SOLO DIVE. CRAY FISHING. PROBABLY LOST REGULATOR FROM MOUTH AND FAILED TO RECAPTURE AND REPLACE IT IN TIME.

SC 96/7

The critical facts in this tragedy are that this inexperienced diver falsified her log book to indicate a greater degree of experience, and that this led to the dive leader to give insufficient thought to close control of the divers he was leading. She and a friend were travelling around Australia on a working holiday. While staying in a hostel they heard of the chance to join a marine science organisation. Anticipating that there might be an opportunity to dive she and several others obtained an appointment. Before being permitted to dive they had to show proof of training, that they had made a certain number of dives and had satisfied one of the staff they were indeed safe divers. She amended her dive log to appear more experienced than she was and this was not noticed when the book was examined. This is not surprising as there is a common assumption that such evidence is true, and it was hindsight which led others to claim the log was so obviously untrue that this should have been so recognised.

The staff member who took the group on a recreational dive to assess their abilities also assumed they were sufficiently experienced to manage a simple dive situation and were capable of watching their contents gauges. He was remiss, but not without cause, in omitting to check her gauge when he checked the others and decided it was time to ascend to the surface, though he had checked it earlier in the dive, assuming her air use would be similar to that of her buddy. He was surprised by the absence of the victim as he began to bring the group up. When he reached the surface the boatman told him that a diver had surfaced a short time before them, waved an arm and then sank. Her mask was off at this time. An initial search failed to locate her but she was later found drowned on the sea bed.

The autopsy report was grossly inadequate, but fortunately a CT scan, which showed conclusive evidence of air embolisation into the heart, was made before the post mortem. It is assumed that she had suddenly found she was in a critical low-air situation and made an emergency ascent which had resulted in pulmonary barotrauma. It was noted as strange that the admission that she had cold water related asthma caused no questioning of her fitness, even though the water in the area was not cold.

Add barristers (there were 6) to an inquest and the dispassionate search for truth becomes a victim. The blame-shifting operation was successful in causing the organisation to tighten its rules and ensure that a stricter check of documents and more careful dive assessment be instituted. However, there may be need to question training standards where monitoring of the contents gauge is not treated as a top priority by pupils. The staff member deputed to take this dive had no special qualification to assess or lead a group, but had that responsibility.

TRAINED. ALTERED LOG BOOK TO SHOW MORE EXPERIENCE. AIR USE INITIALLY SIMILAR TO OTHERS. BECAME LOW ON AIR MORE RAPIDLY THAN THE OTHERS. SUDDEN ASCENT WITHOUT WARNING TO OTHERS. AIR EMBOLISM DEATH. CAGE.

SC 96/8

Because the visibility was so poor at the planned dive site the divers were offered a credit for a future dive by the

dive organiser. Despite these conditions the victim and his buddy dived, unlike the other divers who had rapidly returned to the dive boat. The second location, a reef area, also presented problems although the visibility was better. The divers were told to swim away from the reef after descending, as there was rough water around it. By accident the victim and his buddy failed to follow this advice so surfaced in the rough water around the reef. The water here was too shallow for the dive boat to reach them and they then made their second critical error. Instead of inflating their buoyancy vests and allowing themselves to be washed over the reef into calmer water they attempted to swim through the surf area to reach the boat. The buddy was successful in reaching an area where the boat could reach him, his friend was not. He was recovered by some of the group and towed to the shore and resuscitation commenced. Although he reached hospital and was intensively treated he died there the next day from cerebral hypoxic damage.

The victim had been trained for 2 years but not dived in the previous 12 months. However he was regarded as being an experienced diver by the dive organiser, the instructor who had trained him. Although the victim had indicated to his buddy about the time of their ascent that he had some leg cramp he had declined any assistance. The buddy inflated his buoyancy vest at the surface but it is not stated whether the victim inflated his. The buddy was very tired when reached and the victim would have been similarly affected. The swim might have been easier underwater rather than at the surface.

TRAINED. EXPERIENCED. SURFACED IN SURF AREA OVER REEF. WATER TOO SHALLOW FOR DIVE BOAT TO REACH. FATIGUE. WATER POWER. NO DIVES FOR 12 MONTHS. MADE MISTAKE IN DIRECTION UNDERWATER SO SURFACED IN SURF ZONE. DELAYED DROWNING DEATH.

SC 96/9

This woman was reasonably experienced, having made 44 dives in a wide variety of places. This was her third time at this location. However she had always previously dived with her husband, an instructor, as her buddy. The planned dive had to be aborted as it was close to a main shipping channel and they were told a large ship was due. The alternative location was a frequently dived area. Because her husband had some pupils she was to dive with two other divers. The plan was for them to descend a shot line to a ledge at 20 m at the mouth of the depression. However unknown to the divers, the line was directly over deeper water, 33 m.

The three divers entered the water before the instructor with his 3 pupils and a divemaster. One buddy descended slowly because he was unfamiliar with his hired equipment and was left behind by the other two. This diver joined the instructor's group when they reached his depth. When the victim and her remaining buddy reached the end of the line they were out of sight of the instructor and his group of divers. They stopped at 22 m, the end of the shot line. The victim was a short distance from her buddy, who was holding the line. When the buddy looked up to try to see if the other buddy was coming visual contact was lost. Finding herself alone, the buddy followed correct procedure and ascended. When all the divers had returned to the boat, the victim's continued absence worried her friends but her husband was so confident of her ability that he remained unworried far longer than anyone else. When he did become worried he made a short dive and then an unsuccessful surface search. Then the alarm was raised and a more organised search initiated. The first, at 20 m, was unsuccessful, but a second one to 30 m located her lying on the sea bed.

Probably there were adverse several factors. She was used to diving buddied with her husband, a diving instructor. She was possibly uncomfortable with the 50 m surface snorkel swim from the boat to the shot line buoy, made necessary by the boat drifting, and her wet suit jacket may have been too tight. She descended without retaining her grip on the line and continued the descent after becoming separated. Other factors were nitrogen narcosis, cold, darkness, and separation. She had adequate air but failed to inflate her buoyancy vest or drop her weight belt. The autopsy showed that at some stage of the dive she had suffered a ruptured right ear drum, which could have caused pain and then cold water induced vertigo. But the full story of what occurred can never be known as she was alone at the time. When last seen she was vertical, a short distance from the line and her buddy, rotating as if checking her situation. She had then continued her descent deeper than the agreed maximum dive depth.

TRAINED. EXPERIENCED. TRIO GROUP. FAILED TO WAIT FOR SLOWEST DURING DESCENT. FATIGUE AFTER SURFACE SNORKEL. POSSIBLY TIGHT WET SUIT. MISPLACED SHOT LINE. FAILED TO HOLD SHOT LINE DURING DESCENT. CONTINUED DESCENT AFTER SEPARATION. NITROGEN NARCOSIS FACTOR. COLD. DARK. SEPARATION. RIGHT EARDRUM RUPTURED. ADEQUATE AIR. FAILED TO INFLATE BUOYANCY VEST OR DROP WEIGHT BELT.

SC 96/10

The dive shop checked their certification when they paid to join the boat dive but omitted to look at their log books to establish their degree of experience. The victim could rightly claim to be an Advanced Diver but he had only made a total of 9 dives in the 2 months since he first started scuba diving. He omitted to tell them that he had run out of air on two of these 9 dives. His friend was open

water trained and had made 13 dives, 6 in the last 6 months, but regarded him as the senior diver because of his additional qualification and his very obvious confidence. There were six divers, plus an instructor, who remained in the boat to act as dive master. The water conditions at the reef were variously described, ranging from "a moderate swell" to "a 2 metre swell", with a minimal current. The visibility was regarded as good for this location. The instructor checked that all had their air turned on before anyone entered the water. The victim and his buddy were the last pair to enter the water. Their descent was slow as they experienced problems equalising their ears.

For this dive the victim was wearing his new buoyancy vest and a hired weight belt (11 kg or 24 lbs). Their contents gauges were showing 50 bar after 10-15 minutes at 32 m and they were approaching the anchor line at about 12 m when the victim suddenly grabbed his buddy's buoyancy vest and tried to suck air from it, then abruptly let go of it and disappeared from the buddy's view. She then decided, correctly, to ascend and reached the surface shortly after him.

The instructor saw a diver surface, followed shortly first by the victim (about 20 m from the boat) and then his buddy. The victim was coughing and failed to answer to calls so the instructor jumped into the water and swam with the Jesus line to these two divers. They grasped the line and were pulled to the boat. The buddy was exhausted when pulled aboard and had lost one fin. Both had positive buoyancy but is it not stated whether either had an inflated buoyancy vest. After the buddy was pulled aboard, the victim, who had been holding onto the boat waiting his turn to board, was noticed to have stopped breathing. He was quickly pulled aboard and resuscitation efforts commenced but he failed to respond. Although he had not spoken after he surfaced he had correctly followed all instructions. It is not stated whether he used either his regulator or snorkel after reaching the surface.

Examination of his equipment explained his action in trying to obtain air from his buddy's BCD as his secondary regulator was fed from his BCD. No faults were found in his equipment except for the fact that the tank was empty. A chest X-ray was performed before commencing the autopsy and this showed the presence of air in both ventricles, the right atrium, the aorta, the portal system, and inferior vena cava. There was no pneumothorax or surgical emphysema but the lung histology showed changes typical of barotrauma. The myocardium showed no ischaemic changes, the most atheroma being in the right coronary artery which had less than 30% occlusion. It is possible that nitrogen narcosis effected his response to monitoring his air supply and led to him becoming out-of-air during his ascent. The mismanaged attempt to buddy breath was a further adverse factor and was due to unfamiliarity with the equipment each diver was using.

"ADVANCED DIVER" AFTER 9 DIVES. OVERCONFIDENCE IN HIS DIVING ABILITY. OUT OF AIR. INAPPROPRIATE ATTEMPT TO BREATH FROM BUDDY'S BCD. THEN SEPARATION AND PANIC ASCENT. FACTOR OF EQUIPMENT DIFFERENCES. POSSIBLE NITROGEN NARCOSIS FACTOR. TWICE OUT-OF-AIR IN NINE DIVES OF TRAINING. OUT-OF-AIR THIS DIVE. INEXPERIENCE. PULMONARY BAROTRAUMA. AIR EMBOLISM. CAGE.

SC 96/11

During the time he was employed at this tuna farm this man received some instruction in the use of hookah (surface supply) diving apparatus. He had no formal training in its use, however. It is probable that he had made 12 dives with this equipment before the day on which the man who had "instructed" him told him to take a scuba set and dive inside the tuna cage with him help to repair the inner net. This was the first time he had ever used scuba. This employee evidently assumed that the victim's hose supply diving was sufficient diving experience for this task.

There was poor visibility inside the net and a "reasonably strong current", so strenuous exertion was required. In addition, the depth (20 m) was the deepest the victim had ever dived. Not surprisingly, his air use was heavy. Unfortunately the equipment gave little advance wanting of the exhaustion of the air until this was nearly complete. The victim approached the other diver, who was using surface supply, who signalled to him to ascend, which he did. However he failed to reach the surface and was found dead on the floor of the cage after an, initially unsuccessful, search.

Examination of his equipment showed there was no remaining air and that there was a leak in the scuba feed inflator button. This would have caused an inflated vest to deflate in about 5 minutes but had no relevance to this death as there was no air available to inflate the vest had this been attempted. The pre-autopsy X-ray clearly demonstrated the presence of air in all the cardiac chambers, and a CT examination showed air in the cerebral vessels, the sub-dural space and jugular veins. There was no pneumothorax. It is probable that he suffered the CAGE during his out-of-air ascent, lost consciousness, then sank back to the net floor of the cage. The buddy, who had told him to dive, was so overcome by what had occurred that he later committed suicide, compounding the tragedy. It is not certain that, had the buddy attempted to buddy breathe, this would have been successful in these circumstances.

FIRST USE OF SCUBA. SOME EXPERIENCE WITH HOOKAH. NO FORMAL TRAINING. WORK DIVE IN POOR VISIBILITY, STRONG CURRENT. HEAVY WORK. OUT OF AIR ASCENT. CAGE.

Discussion

Only two of the snorkel using fatalities (BH 96/4 and BH 96/11) were breath-hold diving, the other nine were swimmers using mask and snorkel, with little if any prior experience. Seven were overseas visitors. The significance of this fact may lie in their lack of swimming experience and hence panic when they experienced some problem. All the victims, except the spear fisherman (BH 96/4), were found floating quietly at the surface, in calm water. Health factors were probably responsible in 4 (BH 96/2, BH 96/3, BH 96/7 and BH 96/11). The only experienced victim suffered a post-hyperventilation blackout resulting in his drowning.

The snorkel should no longer be regarded as a totally safe piece of equipment and its use incapable of placing its user in danger. It must be recognised that in a crisis a person's mind may become so focussed on the immediate problem that it fails to allow any consideration of the alternative options for managing the situation. In respect to these fatalities it may be postulated that the unfortunate victims were so consumed with their problem of managing the entry of water through the snorkel that they never thought to remove it and face the situation by becoming simple swimmers once more. It is doubly tragic that, in at least 3 cases, the victims could have simply stood up in the shallow water.

There were 11 scuba diver fatalities identified and CAGE was identified as the critical terminal factor in 6 of them. There were several adverse factors which appeared to influence scuba fatalities, among them inexperience (Table 1), a tight wet or dry suit and strong currents. As usual, running short of air was critical in some cases. In one case an instructor was unfortunate enough to lose a pupil while in close contact and managing his ascent, this indicates the difficulty of even a trained person controlling the actions of others underwater. Inexperience, or lack of recent diving experience, was noted in seven (SC 96/1, SC 96/4, SC 96/5, SC 96/7, SC 96/8, SC 96/10 and SC 96/11). In case SC 96/9 the victim had experience of a range of diving situations but always was dependent on an extensively experienced buddy. In the fatal dive there were several new experiences, a trio group with unknown partners, a tiring surface swim to reach the shot line buoy, descent beyond the end of the line into featureless water, then isolation and nitrogen narcosis. A lethal cocktail of factors.

"Advanced Diver" is a much misunderstood term which does not mean what it suggests. It does not indicate an experienced (advanced) diver, for the Advanced Diver Certificate only means the diver has completed 9 dives under supervision. In case SC 96/5 the diver was taking an "advanced diver" course after only 4 previous scuba dives. He apparently panicked on reaching the sea bed and

TABLE 1
SCUBA DIVING DEATHS AND EXPERIENCE

Inexperienced

Case	Cause of death	Other factors
SC 96/1	Drowned	Adequate air. No dives for 5 years. Dive abandoned due to water filling mask. Rough water. Died during surface swim after separation.
SC 96/4	CAGE	Out of air during low air ascent. 12th dive. Separation during ascent.
SC 96/5	CAGE	5th dive. 1st dive on "advanced diver" course. Distress, possibly due to angina, led instructor to commence ascent with victim who removed his regulator during the ascent and would not replace it. Panic ?
SC 96/7	CAGE	Faked log book entries. Out of air solo ascent.
SC 96/10	CAGE	Advanced diver certification. On 10th dive. Deep dive. Out of air during ascent. Attempted buddy breathing.
SC 96/11	CAGE	Work dive. Some hookah experience. 1st scuba dive. Out of air solo ascent.

Experienced

Case	Cause of death	Other factors
SC 96/2	CAGE	Psychological and psychiatric problems. Deep dive aborted at 5 m. Solo dive after that. Low remaining air.
SC 96/3	Drowned	Negatively buoyant on surface. Strong current. Technical dive. Seemed to be orally inflating buoyancy compensator when he lost his grip and sank.
SC 96/6	Drowned	Solo dive, cray fishing. Legs under a ledge when found. Regulator out of mouth. Adequate air remaining.
SC 96/8	Drowned	No dives for 12 months. Navigational error underwater so surfaced in rough water too shallow for the dive boat. Adequate air remaining.
SC 96/9	Drowned	Trio separated during descent. Shot line incorrectly placed. Solo dive. Adequate air remaining.

suffered a fatal CAGE during a controlled ascent. In case SC 96/10 the victim, certified as an "advanced diver" had managed to run out of air twice during nine training dives. This warning of incompetent air management was not schooled out of him. On the fatal dive he made the same mistake again and this time unfortunately failed to survive. His attempt to "buddy breath" from his buddy's BCD implies that during training he had heard of this unusual procedure, one likely to be of little practical value compared with closer attention to his contents gauge.

There were 3 experienced divers in this series of fatalities. In case SC 96/3 it was the apparent failure to connect his BCD inflation system before entering the water which led to the need for him to attempt (unsuccessfully) to inflate his vest orally. This, combined with the strong surface current, negative surface buoyancy from wearing excessive weights, a possibly tight neck seal to his dry suit and losing of his hold on the mermaid line led to his death. In case SC 96/6 the victim was solo and the presumption is that he was concentrating so much on catching a crayfish that when he lost his regulator from his mouth he was in a position which prevented him from putting it back in his mouth and from making an immediate ascent. Case SC 96/9 is discussed above.

Health factors noted were temporary (fatigue SC 96/9, leg cramp SC 96/8), potential (the cold water asthma history in SC 96/7, personality factors in SC 96/2), or actual but unknown to the victim (myopathy SC 96/1). Depths of 30 m or greater bring nitrogen narcosis into consideration as a factor affecting the responses of the diver to his or her situation, while strong currents affected the course of the dives in cases SC 96/2 and SC 96/3.

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 the police when they are approached for assistance.

Readers are asked to assist this safety project (PROJECT STICKYBEAK) by contacting the author with information, however tenuous, of serious or fatal incidents involving persons using a snorkel, scuba, hose supply or any form of rebreather apparatus.

All communications are treated as being medically confidential. The information is essential if such incidents are to be identified and avoided in future. Please write to Dr D G Walker, PO Box 120 Narrabeen, NSW 2101.

Dr D G Walker is a foundation member of SPUMS. He has been gathering statistics about diving accidents and deaths since the early 1970s. He is the author of REPORT ON AUSTRALIAN DIVING-RELATED DEATHS 1972-1993 which was published in 1998. His address is PO Box 120, Narrabeen, New South Wales 2101, Australia. Fax + 61-02-9970-6004

SNORKEL DIVING A REVIEW

Carl Edmonds

Key Words

Barotrauma, breathhold, deaths, decompression illness, hyperventilation, hypoxia, recreational diving, unconscious.

Introduction

Snorkel diving is related to breath-hold diving and free diving.

The earliest evidence of breath-hold diving is attributed to shell divers, around 4500 B.C.

Traditional breath-hold divers include: the female shell divers of Japan (Ama) and Korea (Hae-Nyo); the sea-men (Katsugi) of Japan; sponge divers of Greece; pearl divers of the Tuamotu archipelago and Bahrain, and the underwater warriors of Xerxes.[1,2]

The abalone and paua divers of the USA and New Zealand and spear fishermen world wide use snorkels to simplify the surface phase of breath-hold diving. Submarine escape tank operators of USA, Europe and Australia have adapted breath-hold diving to modern applications.

The number of professional breath-hold divers of Korea and Japan have remained steady at about 20,000. The pearl divers of the Tuamotu archipelago, the Middle East and the Torres Strait, as well as the sponge divers of Greece, no longer have a viable industry. The abalone and paua divers have remained fairly constant, probably only a few hundred, because of the dwindling supply of this natural resource in shallow, accessible waters.

Compressed air diving, including scuba and hookah (surface supply from a compressor), have eroded the occupational activities associated in the past with breath-hold diving.

The recreational snorkellers of Australia are now a major part of the tourist industry of the Great Barrier Reef. Similar explosions of population are seen in the Caribbean, IndoPacific Islands and the Mediterranean.

Recreational snorkelling has become one of the most widely embraced sports of the latter part of the 20th century but the risks associated with this type of diving are not well documented.

A small group of adventurers has extended the depths, as well as the techniques and parameters of deep breath-hold diving. Sometimes the descents and/or ascents are assisted by weights, floats etc. Sometimes the breathing gases or techniques are modified to extend the depth/duration envelope of breath-hold diving. Currently the depth limit exceeds 150 metres and the duration can be extended by various methods to over 10 minutes. With this complexity comes added risks, outside the scope of this review.

Fatality statistics

The mortality and morbidity incidence in recreational snorkel swimming/diving is unknown, as is the number of the snorkelling population. There is little pertinent data available in the general medical literature, even on the deaths from this activity.

A series of 60 Australian snorkelling deaths (1987-96)[3] were compared to a previous series of 90 Australian deaths (<1987).[4] In the 1987-96 decade the average age had risen from 30 to 45 years. Females had increased from 2% to 25% and were mostly in the drowning group. Spear fishing had reduced from 73% to 25% and by now most of the snorkellers were recreational swimmers engaged in organised boat operations.

The three major causes of death in the latest survey were drowning (45%), cardiac (30%) and hypoxia from breath-holding after hyperventilation and/or ascent, producing unconsciousness and drowning (20%). The incidences in the earlier survey were 52%, 3% and 18% respectively.

Some deaths resulted from marine animals and trauma. Other causes, not obvious from these figures, included epilepsy, cerebro-vascular accidents, asthma, aspiration of vomitus, and entanglement. All these were relatively less frequent.

Recreational snorkellers were mainly surface swimmers who occasionally dived. The breath-hold divers were often spear fishermen and collectors of shellfish.

Other characteristics of the 1987-96 survey, which did not differ significantly from those of the previous one, showed that; only 7% had an allocated “buddy diver” accompanying them at the time, 7% were separated by the incident, 47% separated prior to the incident, 40% were solo snorkelling from the start.

This is reflected in the absence of rescue attempts in 42%. In 58% a rescue attempt was made, usually by an unassociated swimmer. Rescue was attempted in under 5 minutes (when resuscitation is feasible) in only 17%, and in 38% it took between 5 and 15 minutes before being initiated.

Many factors contributed to the three major causes of death.

Tourists were over-represented in both the drowning and the cardiac groups. Inexperience, medical and physical unfitness, equipment and environmental factors contributed to the deaths in these two groups.

DROWNING

Drowning cases were frequently inexperienced, medically or physically unfit, inadequately equipped (often without fins, to assist in propulsion) thereby increasing the danger of adverse sea conditions. Supervision was inadequate and therefore rescue and resuscitation were delayed.

It occurred frequently in non-English speaking tourists who may not have understood either the documentation required by commercial boat operators, or the safety instructions from snorkelling supervisors.

CARDIAC

Cardiac deaths were analogous to those in middle aged males while scuba diving.[2,5] They died quietly, usually on the surface. There are many aquatic factors that can precipitate a cardiac event, when superimposed on an underlying cardiac disorder, which was often evident in the medical history.

Inexperience, poor physical fitness, poor swimming ability and the absence of efficient propulsion, all increased the effort required for snorkelling.

The extreme difficulty of achieving adequate resuscitation with this presentation, has been noted with scuba.[6]

HYPOXIA

Hypoxia from hyperventilation and/or ascent,[3,7-10] during breath-hold diving, occurs predominantly in spear

fishermen. The hypoxia develops because of the increasing breath-hold time possible after hyperventilation, due to the production of hypocapnoea. With the hyperventilation induced hypocapnoea, there is no proportional increase in oxygen carrying capacity, and therefore these breath-hold divers are able to extend the "breaking point" to such a degree that hypoxia and therefore unconsciousness may result. In these cases there is little or no warning.

Hypoxia is aggravated during ascent because of the expansion of gas (Boyle's Law) in the breath-hold diver's lungs, reducing the partial pressure of oxygen and increasing the likelihood of hypoxic unconsciousness.

This is a younger, fitter, group of divers, very experienced and with good equipment, undertaking a hazardous diving technique which should be discouraged.

TABLE 1

PROFILES OF DEATHS

Cause of death	Drowning N=27	Cardiac N=18	Hypoxia N=12
Male: Female	15: 12	15: 3	12: 0
Average Age (SD)	44.7 (18.7)	55.6 (14.6)	35.2 (14.9)
Site of death	Surface	Surface	Depth/ascent
Equipment	Inadequate	Inadequate	Complete
Nationality	48% Foreign esp. Japanese	50% Foreign esp. USA	100% Australian
Experienced	30%	50%	100%
Buoyancy	Positive	Positive	Negative

Marine environments

Like all other divers,[2] breath-hold divers are susceptible to the hazards of the marine environment. These include injuries from marine animals, infections and envenomations. They include exposure to water temperatures less than thermo-neutral (35°C) as well as the various drowning syndromes, including salt water aspiration and near-drowning. Motion sickness is a common problem, as is trauma (ocean currents, rocks, boats etc.) and entrapment.

Equipment problems

These hazards include a variety of problems due to the actual equipment being worn by the free diver, i.e. mask, snorkel, fins etc., together with the problems of entrapment, the use of spear guns, floats, boats etc. They are no different in principal from those encountered by scuba divers, but the disadvantage for the free diver is that a plentiful supply of air is not available to him. One of the increasing dangers is entrapment and entanglement in lines (floats, spears, etc.). Some of the modern filament fishing lines are not able to be snapped nor cut by a knife. A scuba diver has much more time available to cope with such difficulties.

Common equipment problems include flooding of the face mask and restriction to snorkel breathing with exertion. The reduction in maximum voluntary ventilation and increase in the work of breathing produces dyspnoea when the respiratory demands are great.

Barotrauma

Barotrauma of descent [2] is more common in free divers than scuba divers, because of the rushed nature of the activity. Free divers have so little time that they have to descend more rapidly and often without much attention to the various symptoms that may be caused by barotrauma. They also undertake more ascents and descents, producing more barotrauma. The areas affected are; face, ears, sinus, dental and the gastrointestinal tract.

Following barotrauma of descent, there is often an associated barotrauma of ascent. This is especially seen with otological, sinus, dental and gastrointestinal barotraumas. Ascent cannot be delayed, nor even slowed, and so the manifestations cannot be diminished, as they are with scuba.

Pulmonary barotrauma of descent (lung squeeze),[11,12] occasionally occurs in breath-hold divers.

With descent, the gas volume in a breath-hold diver's lungs will contract in accordance with Boyle's Law. Thus a diver with a total lung volume of 6 litres (and residual volume of 1.5 litres) on the surface (1 ATA) will be able to descend to 4 ATA (30 m) before the lung volumes will equate with the residual i.e. ($P_1 V_1 = P_2 V_2$), 6 x 1 = 1.5 x 4.

Initially it was believed that further descent would lead to lung pathology (haemorrhages, oedema etc.). In practice this is avoided by the negative pulmonary pressure resulting in engorgement of the pulmonary vessels. This reduces the residual volume, replacing air spaces with intravascular blood from the periphery.

Breath-hold descents, without inducing "lung squeeze", have been undertaken to depths in excess of 150 m.

Decompression sickness

Decompression sickness has also been postulated as a result of intensive free diving.[13-16]

Cross described an illness called Taravana (tara = to fall, vana = crazily), in the pearl divers of the Tuamotu archipelago.[13,14] The dives were to 30-40 m, lasting 1.5 to 2.5 minutes each, over a 7 hour period. The illness, which was characterised by vertigo, nausea, paresis, unconsciousness or death, could be due to decompression sickness in some of the cases. Perusal of the original cases would indicate that many could have been due to a variety of other disorders, e.g. inner ear barotrauma, salt water aspiration, near drowning causing hypoxic encephalopathy and drowning.

The reason decompression sickness can develop with breath-hold diving is that the nitrogen pressure in the lungs increases with depth, and with the greater depths there is a greater nitrogen partial pressure, with nitrogen diffusing from the lungs into the bloodstream and thence to the tissues. If the surface interval is inadequate to eliminate this nitrogen, or if bubbles develop due to the rapid ascent, then it will accumulate with repeated dives throughout the day.

Paulev, a Danish submarine escape tank safety diver in the Norwegian Navy, performed 60 breath-hold dives to 20 m in 5 hours, each lasting about 2.5 minutes with surface intervals of less than 2 minutes. He developed symptoms consistent with decompression sickness.[15] Other submarine escape instructors have suffered similar problems, in both Norway and Australia.[16]

Hypoxic blackout

This is sometimes called breath-hold syncope or "shallow water blackout". As the latter term was first used in 1944 to describe loss of consciousness using closed circuit diving apparatus, it is best avoided in the breath-holding context. "Hypoxic blackout" is a reasonable alternative.

There are two causes for this disorder,[7-10] hyperventilation and ascent, and, as they may occur concurrently, they are often confused. The hyperventilation effect is independent of depth, and may be encountered in 1 m deep swimming pools, often by children trying to swim greater distances under water.

Divers who train to extend their breath-hold time and also dive deep (such as in free diving competitors, spear fishing etc.) risk hypoxia of ascent, with loss of consciousness and subsequent drowning.

With hypoxia there is little or no warning of impending unconsciousness. With increased experience the breath-hold diver can delay the need to inhale by various techniques, without improving his oxygen status. Breath-hold time can be extended (but not with increased safety) by feet first descent, training (adaptation), swallowing, inhaling against a closed glottis, diaphragmatic contractions etc.[11]

One way of avoiding this hypoxia is to inhale 100% oxygen prior to the breath-hold.

HYPERVENTILATION AND HYPOXIA

Craig observed that swimmers who hyperventilated could stay longer underwater, but risk losing consciousness with little or no warning.[7-9] They were often competing, against others or themselves, and often exercising. The hyperventilation extended their breathholding time, because it washed out a large amount of CO_2 from the lungs, often reducing arterial CO_2 to half the normal levels.

The build-up of CO_2 is the main stimulus compelling the swimmer to surface and breath. After hyperventilating it takes much longer for this level (the "breaking point") to be achieved.

The arterial O_2 pressure drops to a level inadequate to sustain consciousness, if breath-hold time is extended and exercise consumes the available oxygen.

One can see the effects of both hyperventilation and exercise in reducing the O_2 level to a dangerous degree, when breath-holding, in Craig's original experiment.[7] Table 2 (page 20) reproduces his results.

When the swimmer is concentrating on some purposeful goal, such as trying to spear a fish, retrieve a catch, or untangle an anchor, then he is more likely to ignore the physiological warning symptoms of an urge to breath (due to the rise in CO_2 level in the blood), and delay the breaking point.

HYPOXIA OF ASCENT

Ascent hypoxia was described first in military divers using gas mixtures, who lost consciousness as they surfaced with low oxygen levels in their rebreathing equipment.[2]

In breath-hold divers, with descent the pressure rises proportionately in the alveoli gases, increasing the

TABLE 2

EFFECTS OF HYPERVENTILATION ON THE BREATH-HOLDING (BH) TIME AND ALVEOLAR GAS PRESSURE AT THE BREAKING POINT IN RESTING AND EXERCISING MAN[7]

MEASUREMENTS	RESTING	
	Without Hyperventilation	With Hyperventilation
BH time (sec)	87	146
End-tidal pCO_2 (mm Hg)		
Before BH	40	21
Breaking point	51	46
End-tidal pO_2 (mm Hg)		
Before BH	103	131
Breaking point	73	58
	EXERCISING	
	Without Hyperventilation	With Hyperventilation
BH time (sec)	62	85
End-tidal pCO_2 (mm Hg)		
Before BH	38	22
Breaking point	54	49
End-tidal pO_2 (mm Hg)		
Before BH	102	130
Breaking point	54	43

available O_2, CO_2 and nitrogen uptake. Some O_2 can be absorbed and utilised, some CO_2 absorbed and buffered, some nitrogen absorbed and deposited in tissues.

Thus if a diver, having 100 mm Hg 0_2 and 40 mm Hg CO_2 in his alveolar gases, was immediately transported to 2 ATA, the lungs would halve their volume (Boyle's Law P1 V1 = P2 V2), the O_2 would be 200 mm Hg, and the CO_2 80 mm Hg. Both would pass into the pulmonary blood circuit, the O_2 to be used and the CO_2 to be buffered. Thus the O_2 and CO_2 pressures in the alveoli would decrease rapidly. By the time they were both back to "normal" levels, with O_2=100 mm Hg and CO_2 = 40 mm Hg, then the diver would appear to be in a satisfactory respiratory status, until he ascended. With an expansion of the lungs to twice their size at depth, the pressures in both gases would halve, i.e. the O_2 would drop to 50 mm Hg (approaching a potentially dangerous hypoxic level) and the CO_2 to 20 mm Hg, if the ascent was immediate.

As ascents do take time, more O_2 will be consumed, extracted from the lungs during the ascent, and the CO_2 would increase towards normal due to the gradient between the pulmonary blood and alveoli. The actual figures would vary with the speed of ascent, the diffusion gradient and rate, and the consumption of O_2.

The drop in O_2 is then able to produce the loss of consciousness, the "syncope" or "blackout", commonly noted amongst spear fishermen. This is now known as hypoxia of ascent. In deeper dives it becomes more likely, and with some very deep dives, the loss of consciousness may occur on the way to the surface, in the top 10 m (probably an explanation for the "seven metre syncope" described by French workers[17]).

OTHERS

Despite the classical causes of hypoxia, as above, probably the commonest cause is the aspiration of salt water, resulting in near drowning and drowning states[3,6].

Cardiac disorders [18-25]

Human breath-hold divers produce a dramatic bradycardia from the diving reflex.[11] It reaches its zenith in 20 to 30 seconds, usually to an equivalent of two-thirds the pre-dive level, but sometimes to less than 10 beats/minute in experienced divers. It bears a linear relationship to the water temperature below 15°C, non-linear above that. The bradycardia might then permit other arrhythmias to develop. The arterial blood pressure seems to increase with the diving reflex in humans.

In humans, unlike most of the diving mammals, free diving is associated with significant cardiac arrhythmias. These can be provoked by common respiratory manoeuvres such as deep inspiration, prolonged inspiration, breath-holding, release of breath-holding, Valsalva manoeuvres etc.[24]

In a study of Korean women divers the incidence of cardiac arrhythmias was 43% in the summer (water temperature 27°C) as compared with 72% in the winter (water temperature 10°C).[18]

There is a high frequency of arrhythmias in association with immersion breath-holding, even without diving. The head-out immersion position increases the workload on the heart, because of the negative pressure effect (the intrapulmonary pressure remains at 1 ATA, while a negative pressure, needed to inhale, is approximately -20 cm H_2O). There is a reduction in the functional residual capacity of the lungs, an increased work of breathing and an increase in the intrathoracic blood volume, with a corresponding dilatation of the heart, and especially the right atrium. The latter may be a major cause of arrhythmias from sinus rhythm. Immersion diuresis and associated loss of sodium may exacerbate cardiac problems. With very deep breath-hold divers about a litre of extra blood can fill the pulmonary circuit and the heart.

The relatively high incidence of cardiac deaths during snorkelling[3] (and scuba diving[5]) activities may be

partly related to the above findings, and partly due to the excessive workload experienced by novice snorkellers, attempting to overcome the influences of panic, adverse tidal currents and a need to keep the head above water.

Pulmonary disorders

The most common lung problem is the aspiration of sea water, producing either the drowning syndromes or provoking asthma in those so inclined.

The changes in lung volumes with the head-out position during immersion have been described above, with the pooling of blood in the thorax, reducing respiratory capability.

Pulmonary oedema[2,11,24] has been described in association with immersion, as have other causes of dyspnoea, including the coronary artery disease, cardiac arrhythmia and cold induced hypertension.

Gastrointestinal problems

Another pressure gradient associated with the head-out immersion, commonly experienced in free divers between dives, is the increased gastro-oesophageal pressure gradient, which increases from 6 mm Hg in air to 16 mm Hg during immersion. This predisposes to gastric reflux, in those with an inadequate oesophageal sphincter.

This also increases the tendency to vomiting which can be aggravated by other factors such as alcohol intake, sea sickness, otological barotrauma, gastrointestinal barotrauma etc.

Medical check list for snorkellers

Unless there are specific medical or physical disorders, medical examinations are not usually required by snorkellers. Because there are organised commercial snorkelling activities, on the Great Barrier Reef and by schools or other organisations, the minimal medical requirement should be a simple questionnaire likely to pick up most major causes of mortality and morbidity from this activity. A typical one is reproduced here as Table 3.

Conclusion

Despite the paucity of medical documentation on this common recreational activity, some information is available. Combined with case histories[4] and consensus views of experts in this field, there probably is enough evidence to base recommendations on safety aspects of this activity. Medical fitness can be indicated by a medical history questionnaire for potential snorkellers.

TABLE 3

MEDICAL CHECK LIST FOR SNORKELLERS

Have you ever had:

1	Any cardiovascular disease? (Heart, blood pressure, blood, others).	YES	NO
2	Any lung disease? (Asthma, wheezing, pneumothorax, TB, others).	YES	NO
3	Any fits, epilepsy, convulsions or blackouts?	YES	NO
4	Any serious disease? (Such as diabetes)	YES	NO
5	Serious ear, sinus or eye disease?	YES	NO
6	Any neurological or psychiatric disease?	YES	NO

Over the last month have you had any:

7	Operations, illnesses or treatment?	YES	NO
8	Drugs or medications?	YES	NO
9	If female, are you pregnant?	YES	NO

Can you

10	Swim 500 metres without flippers?	NO	YES
11	Swim 200 metres in 5 min or less, without flippers?	NO	YES
12	Equalise your ears when diving or flying?	NO	YES

NAME: (If under 16 years, guardian to sign)

DOB:

ADDRESS:

Note: If the candidate indicates an answer in the left hand column, then medical assessment and advice is required before snorkelling is undertaken.

A standard of physical and aquatic fitness is a reasonable requirement. Buddy responsibility should be encouraged and adequate supervision available, especially if the snorkeller is not experienced in the existing conditions. Supervision is essential during commercial diving operations.With the latter, multilingual facilities may be required.

Hyperventilation should be discouraged as a prelude to breath-hold diving.

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THE WORLD AS IT IS

OZTEK99 DIVING TECHNOLOGIES AND REBREATHER FORUM A NON-MEDICAL REVIEW

Lynn Taylor

Key Words

Decompression illness, equipment, meeting, mixed gas, rebreathers.

Introduction

On April 24th and 25th 1999 the inaugural OzTeK Diving Technologies Conference and Rebreather Forum was held in Sydney, Australia. The 1999 Asia-Pacific TDI (Technical Diving International) Members Forum had been held over the two previous days, so some attendees had 4 enthralling days of presentations and workshops from some of the World's leading Technical Diving identities.

Opening address and history of rebreathers

The convener, Richard Taylor, (TDI Australia and New Zealand) welcomed us to the conference on Saturday morning. Bret Gilliam (President TDI) then set the scene with a keynote speech reviewing the past, present and future of technical diving. This included a look at some video footage taking advantage of the relative silence of a rebreather, enabling the cameraman to get close in amongst a school of hammerhead sharks. Adding reality and entertainment to the scene was the fact that the microphone had picked up the adrenaline-mediated increase in heart rate of the cameraman as the sharks swam so unbelievably close! Bob Ramsay of the Diving Historical Society Australia and South East Asia (DHS ASEA) followed, with a history of rebreathers. He informed us that the first documentation of "rebreather" use dated as far back as 1624, with a description of a submarine made out of a barrel, and containing a special pot of unknown liquid, being used in the River Thames. The first design of an oxygen rebreather was first documented in 1778.

Decompression theory and application

From a historical overview, the morning evolved into decompression theory and application starting with Chris Parrett (author of the Abyss Advanced Dive Planner) discussing the micro-bubble theory and its relevance to technical diving requiring decompression stops. This theory supposes that tiny bubble "nuclei" (microscopic pockets of gas) are naturally present within our bodies and that excess gas diffuses into these nuclei, expanding them and so creating bubbles. Once bubbles form they may join together and form larger bubbles and may distort other tissues. Bubbles can become trapped and put pressure on nerves, damage tissues or block the blood supply to vital organs. As the ambient pressure is reduced during ascent, the bubbles grow in accordance with Boyle's Law (*P*ressure x *V*olume = *k* constant). In addition, since the pressure inside the bubble will always be roughly equivalent to ambient pressure, any gas which remains dissolved in tissue at greater than ambient pressure (from a previous depth) will tend to move into the (lower pressure) bubble, causing it to expand further causing more damage.

It has been suggested that these micro-bubbles can be crushed by high pressure. This is the basis of the controversial theory behind making an initial deep dive for a few minutes, before the day's diving activities, to crush the micro-bubbles. It is thought that the micro-bubbles take somewhere between hours and days to regenerate so, theoretically, regular diving every few days may have beneficial effects by keeping micro-bubbles crushed.

Of interest to technical divers, Chris presented evidence to show that on decompression dives, deep, short (1-3 minute) stops have advantages. The long ascent to the first stop, in traditional decompression profiles, because of its steep gas to ambient pressure gradient, produces many bubbles. Shorter, deeper stops reduce this pressure gradient and so theoretically reduce the potential for bubble formation. Also eliminating bubbles formed early decompression reduces overall decompression time.

Chris translated the micro-bubble theory into general practical safe diving advice, which applies to both technical and recreational divers. He noted that Doppler monitoring of divers has revealed that the number of bubbles peaks approximately 30-60 minutes after surfacing. With this in mind, Chris suggested that repetitive dives within this window of maximal bubble formation (30-120 minutes after a dive) should be avoided. For the same reason, multiple surface ascents within a dive should also be avoided. Other practical applications of the theory include making a slow ascent of 10 m/minute and making a stop in the 3-6 m range on all dives. Diving at altitude requires careful consideration as bubble excitation and bubble growth rates are enhanced by a decrease in ambient pressure. In other words, bubbles grow more easily at altitude and can have greater impact on the diver.

Chris pointed out that the micro-bubble model has not been validated and is sometimes in conflict with the tissue-based model. However early results have been encouraging. Chris also gave participants a chance to win a copy of the Abyss98 Advanced Dive Planning software!

In-water recompression

After lunch there was a very informative forum on in-water recompression (IWR) with three eminent speakers, Dr Ann Kristovitch (Medical Officer), Richard Pyle (Deep Reef Explorer) and Dr Carl Edmonds (Director, Diving Medical Centre). It is accepted within the diving medical community that, in general, IWR should not be attempted as it carries inherent risks. However, in certain circumstances, such as in remote areas or after failed decompression stops in deep technical diving, in-water recompression may be the best, or the only, immediate option. Three important points were emphasised. The correct equipment, including a full-face mask for the delivery of 100% oxygen, must be available for safe IWR. In the case of deep technical divers with missed decompression stops, immediate treatment is essential. Following IWR, a diver should be transported to a medical recompression facility as soon as possible. Various methods of IWR were discussed.

Medical aspects of closed circuit rebreathers

Then Dr Robyn Walker (Royal Australian Navy) enlightened us about medical problems with rebreathers. The main take home message was the importance of checking and re-checking that the level of oxygen is as planned before starting the dive. When oxygen is breathed at partial pressures of greater than 1.4 bar (ATA) the degree of central nervous system toxicity rapidly increases. Unless one is wearing a full-face mask acute oxygen toxicity under water is nearly always fatal. Oxygen toxicity causes fitting without warning, resulting in the diver losing the regulator. The respiratory muscles go into spasm and the diver stops breathing. When respiration restarts the diver will inhale water and drown. Often the first signs of oxygen toxicity are recognised by others observing slightly unusual behaviour, as the diver often does not recognise the subtle indications such as nausea, light headedness, ringing in the ears, a sense of impending doom, sweating and pallor. She also touched on CO_2 toxicity, caustic cocktails and dilution hypoxia.

Cave diving in Australia

After all this fascinating information, I was pleased to sit back, relax and watch Neil Vincent show us wonderful pictures of his cave diving exploits in Australia. Fantastic water clarity and rock formations. Although I do not think you will catch me diving down through the "birth canal" (in McCavity Cave at Wellington, NSW) and waiting for someone to push my scuba gear after me!

The evening session proved to be both stimulating and entertaining with three presentations. Richard Pyle talked about deep reef explorations and researching new species of fish using the CIS-Lunar MkV rebreather around the Hawaiian Islands at depths of 75-150 m. Jim Bowden gave us an insight into the tremendous physical and psychological demands of the Deep Project in the jungles of Mexico where exploration has led to diving to nearly 300 m (1,000 ft). Finally Nuno Gomes took us through the meticulous planning and execution of his world record scuba dive to 282 m at altitude in South Africa! The audience was mesmerised by the sheer dedication of these men, though there was a teaser thrown out, that no woman has yet dived to beyond 180 m (600 feet).

Diving with closed circuit rebreathers

On Sunday Peter Readey (CABA/Steam Machines) enlightened us with the unexpected things that one can do on your first dive using a rebreather. For example if you want to swim above an obstacle, in traditional scuba you would inhale but if you do this with a rebreather you will crash head long into it! The volume in your lungs and the counter-lung exchange, but remain constant. Also, a gentle reminder was not to leave the mouthpiece in the open position at the surface. This lets the rebreather bag deflate and you will sink!

Treating decompression illness in mixed gas divers

Dr Simon Mitchell, a Specialist in Diving and Hyperbaric Medicine, has left the Royal New Zealand Navy and is now the Medical Director of the Wesley Centre for Hyperbaric Medicine in Brisbane. He briefly reminded us of the mechanisms and manifestations of decompression illness (DCI) and discussed the relative advantages and disadvantages of using helium and/or nitrogen as the inert portion of the diver's breathing gas. His attention then focussed on the treatment of DCI in technical (mixed-gas) divers. The emphasis here was on the divers being prepared and having a plan of action, which can be implemented immediately if the need arises.

DCI resulting from deep technical diving is often neurological DCI with early onset of symptoms and a very rapid deterioration. The window of opportunity to help the situation is small. Involving the nearest recompression facility so they can be on stand-by is an important part of the planning process, as is having correct emergency equipment on hand at the dive site. Dr Mitchell's recommended hierarchy of intervention options in rapidly progressive DCI are:

1 Immediate (in less than 30 minutes) recompression in an appropriate recompression chamber,
2 In-water recompression if done early and with the proper equipment,
3 First aid at the site with evacuation to a recompression chamber.

It is very important to give 100% oxygen immediately in all options.

Controversy surrounds the administration of aspirin in suspected DCI. Gas bubbles in the blood can damage the vessel lining and the body produces platelets, which aggregate to help mend the damage. A consequence of this natural response is that platelets can also stick to the bubbles, so making them more likely to cause a blockage and facilitate the immune response and contributing further to DCI. Aspirin reduces platelet aggregation and theoretically this may help reduce some of the effects of DCI. However, Dr Mitchell pointed out that there is no data, in either animals or humans, which suggests aspirin administration improves the outcome in DCI. Moreover, aspirin may exacerbate any haemorrhage into the spinal cord occurring in the pathology of spinal DCI. The weight of evidence at present, in his opinion, favours not giving aspirin to victims of DCI.

He then addressed some of the logistical concerns in treating technical divers in a recompression chamber. Treatment in a recompression chamber is likely to involve recompression to 18 msw and breathing 100% O_2. Deep technical divers may need to be taken deeper that 18 m to control the bubbles more quickly, but the risks associated with oxygen toxicity mean that 100% O_2 cannot be administered beyond this depth. So a diluent gas must be added with deeper treatments and the dilemma of whether to use helium or nitrogen was discussed.

The basic take home message was “the earlier the treatment the better the results” but sadly, not everyone will have a complete recovery. Dr Mitchell concluded his presentation with his usual panache and entertained the audience with a couple of stories from his navy days.

CO_2 absorber design

The later morning sessions included a look at the Royal Australian Navy (RAN) experiences of rebreather CO_2 scrubber design faults by Dr Carl Edmonds and a review of closed-circuit rebreather failures by John Pennefather (RAN Submarine and Underwater Medicine Unit).

Rebreather try dives

After lunch we could try out a rebreather in the pool! It was $20 well spent. One could chose from Dräger Dolphin and Dräger Ray Rebreathers, Prism, Inspiration, Cis-Lunar, Halcyon, Steam Machine 1600’s and more. I had my first dive with a semi-closed circuit Dräger Dolphin. After getting used to the slight resistance on exhalation, and all the extra weight I needed, I descended to the deep end of the pool and swam around in almost silence. After a while I stopped and observed the scene. It was like a dream, observing. I could not be diving, there were no blasts of bubbles. Amazing. I definitely want to try one in the sea.

There were also presentations on the Wakulla 2 Project (John Vanderleest, one of the Australian divers on the team), Diving the World War 1 Australian submarine AE2 in Turkey (Dr Mark Spencer, the AE2 Project Leader), Doppler Monitoring of bubbles in divers after decompression (Dr Akin Toklu of the AE2 Project Turkish Support Team), and Diving the Atlanta, Britannic and Saratoga (photographer and expedition leader Kevin Denlay).

Throughout the conference, equipment seminars and rebreather forums were taking place in an adjacent room. If only I could have gone to everything!

All in all, an excellent 2 days. Congratulations to Richard Taylor (TDI Australia and New Zealand) for putting together such an excellent and informative program and to the exhibitors who made it all possible.

Lynn Taylor is a PADI IDC Staff Instructor and a DAN O_2 instructor. She came to New Zealand, from England, in 1994 and soon found a passion for diving. Her interests in the technical and medical aspects of diving have stemmed from her science and research background, BSc and PhD, and hence her interest in OzTeK. Her address is 26 Barker Rise, Browns Bay, Auckland, New Zealand. Telephone + 64-9-367-2948. Fax +64-9-367-2500. E-mail ltt21040@GlaxoWellcome.co.uk .

SUB-SPECIALTY CERTIFICATION IN UNDERSEA AND HYPERBARIC MEDICINE

The first examination for certification in Undersea and Hyperbaric Medicine (UHM) was held on 8 November 1999 by the American Board of Preventive Medicine (ABPM), which plans to offer this exam on a regular basis. The requirements to sit the examination may be met either through completion of an approved fellowship in UHM after primary board certification. Or, starting in 2003, through a combination of basic training and practical experience. The applicant must have current certification from one of the 24 Member Boards of the American Board of Medical Specialties. Detailed requirements, application forms and examination content outline are available on the ABPM’s web site at www.abprevmed.org .

Key Words

Hyperbaric oxygen, qualifications, underwater medicine.

SPUMS NOTICES

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.

AWARDS PRESENTED AT THE HTNA SCIENTIFIC MEETING 1999

The Hyperbaric Technicians and Nurses Association (HTNA) Annual Scientific Meeting was held at the Stamford Plaza Hotel, Adelaide, 25-28 August 1999. The meeting brought together representatives from all Australian and New Zealand Hyperbaric Medicine Units along with the invited speakers, Dr Caroline Fife, President of the Undersea and Hyperbaric Medical Society, Dr Paul Sheffield and Mr Tom Workman.

SPUMS award

SPUMS presented the first of what will become an annual award for the most outstanding original paper presented by a HTNA member at the meeting.

Papers by Kathryn Borer and Mike Dawe received a special mention with the prize (Eric Kindwall's Textbook of Hyperbaric Medicine) being awarded to Greg Melbourne of the Prince of Wales Diving and Hyperbaric Medicine Unit for his paper titled "Course accreditation for hyperbaric nurse attendants".

Awards to SPUMS members

During the conference dinner, John Lippmann, on behalf of DAN SEAP, presented two special awards to SPUMS members in recognition of their contributions to diving safety.

Dr John Knight received his award for his active pursuit of diving safety during his many years of association with SPUMS and in more recent times as the Editor of the SPUMS Journal.

Dr Douglas Walker, a Foundation Member of SPUMS, was also recognised by DAN SEAP for his contribution to diving safety through his dedicated collection and publication of diving accident data over many years.

Congratulations to both recipients for this highly deserved recognition.

Robyn Walker
President of SPUMS

Key Words
Meeting.

PRESIDENT'S REPORT FOR 1999

Thank you for attending the 27th Annual Scientific Meeting of our Society, particularly those attending for their first time. I would especially like to thank and acknowledge the guest speakers, Alf Brubakk and Richard Moon. I would also like to thank Greta Bolstad from EUBS for again supporting our meeting with her attendance.

I would like to congratulate Robyn Walker on her elevation to the Presidency of our Society, thus ending my three terms as President. I would like to thank the various committee members over that time for their support and the membership at large for accepting me (and putting up with me) over this time.

I now look forward to arguably the best position in our Society, the Past-President's position. My elevation to Past-President now retires Professor Des Gorman from that Committee position. I wish to acknowledge the many years of commitment to the society by Des Gorman. Unfortunately Des was unable to attend this meeting due to commitments with the University of Auckland.

During the last twelve months there have been a number of matters worthy of special comment.

1 The SPUMS Website (www.SPUMS.org.au) has been opened. It is a significant mechanism for spreading information about our Society and information on diving medicine. It a reference point for members wishing for information about our Scientific Meetings. It is also becoming a useful resource for new members and delegates at ASMs.

2 The SPUMS Grant. In future SPUMS will fund a grant, to be awarded where appropriate, to a diving medical researcher, to present an approved research paper at our Annual Scientific Meeting. Eligibility and amount will be determined by the SPUMS Education Officer in conjunction with the SPUMS Executive. Anyone wishing to apply should contact our education Officer, David Griffiths.

3 SPUMS Stickers. The new SPUMS stickers, promoting our society and the DES telephone number, have been available to members at this meeting. Stickers will be distributed with the June 1999 Journal. Please stick them somewhere prominent.

In 1999 we will be trialing the appointment of an Administrative Officer to perform many of the day to day duties of the Society. Steve Goble, from the Royal Adelaide Hospital, who is already working to maintain the Diving Doctor List, will be performing these duties. We hope this will improve our service to the membership and our response time to membership inquiries and other matters. The SPUMS membership database has been upgraded and updated, Steve Goble will maintain this and the other day to day duties, on a part-time basis. This should also ensure that future changes in the SPUMS executive will not interfere with the day to day operations of the Society.

Later in this meeting we will be considering changes to our constitution. These are entirely procedural changes, largely to correct errors, omissions and changes. I will summarise the important aspects.

Firstly, the Australian and New Zealand Hyperbaric Medicine Group is a sub-committee of SPUMS. That has been the case for some time, but it has never been documented in our constitution, and it is proposed now that it should be.

Secondly, the members of the Australian and New Zealand Hyperbaric Medicine Group must be members of SPUMS. This follows on from the first.

The Chairman of the ANZHMG will have a position on the SPUMS Executive. There are some other changes in the composition of the committee composition to incorporate that.

There is to be a change to the election of the Editor and the Public Officer. In the history of the Society there has never been an election for the Editor. It is proposed that rather than elect the Editor, the Committee will appoint the Editor, and in fact, as the Editor is an employed position of the Society, that is a better relationship to have with the Editor.

We also propose that rather than elect a Public Officer, the Public Officer be appointed. That is largely because we cannot have an open election for a Public Officer. As we are an association incorporated in Victoria, the Public Officer has to reside in Victoria. The reason that we are an incorporated Society is that it gives our Committee members protection from legal action from the public and members.

There are number of other minor changes in the constitution wording to accommodate those changes. We will formally, in the constitution, document the Board of Censors of the Society, and have clearly established who the Board of Censors are. They are in fact, the Education Officer, David Griffiths, the President of the Society and the Director of a Hyperbaric Unit in Australia or New Zealand.

1999 also sees SPUMS formalising its relationship with ANZHMG. In the future the Chairman of ANZHMG will be on the SPUMS Executive and be the official spokesperson on Hyperbaric Medicine matters. The SPUMS President and Secretary will remain the spokespersons on Diving Medical matters.

I look forward to seeing many of you in Fiji in 2000, where the theme will be "Diving Medicine in the New Millennium".

Finally, thank you all for the three years I have enjoyed as President of SPUMS. I hope that SPUMS is a little better at the end of my term and continues to improve under Robyn Walker, with increasing membership, particularly in Europe, USA and Asia. When you return home please encourage your colleagues to join our Society, and to attend our Scientific Meetings.

Guy Williams
President of SPUMS

SECRETARY'S REPORT FOR 1999

Now, in May 1999, the membership database has been reformatted. The number of members on the database is 1,272. Of these, 860 have renewed and are financial members. We have 300 members who are unfinancial since the beginning of this year, who we hope will renew. There are also 112 unfinancial members from the beginning of last year, who are probably not going to renew but are still on the database.

The currently financial membership breakdown is, 830 in Australia, 113 in New Zealand and 225 from overseas. Of the overseas members, approximately 100 are from North America, 100 in Europe and 25 from South East Asia and the Pacific.

The past

This is a quick overview of my time as Secretary, which is 1993 to the present. I was recruited in Port Douglas, at my very first SPUMS meeting, by John Williamson in 1992. I was nominated for and elected Secretary in Palau, at the AGM, in 1993.

Since then, SPUMS has really changed as to how we maintain membership records and our technology. I initially started doing the secretarial duties on my home computer, which was IBM. The Treasurer used a small Macintosh to maintain the database, and she did all the financial records on paper with a cash book. All the correspondence with the committee was by fax and telephone.

In the transition period between Treasurers, in 1996, we bought two Toshiba notebook computers, one for the Treasurer and one for the Secretary. We converted the database to IBM and it was put into Microsoft Access. However the financial records were still on paper. The Committee started to communicate tentatively by e-mail, although still using a lot of fax and phone. Now the financial data is in an electronic data package, the database has been reformatted with increased functionality . This year renewals were made easier for us to handle by printing our current data on the back of the renewal, so that members could ensure that we had the correct information to update our records efficiently. This will lead to increased efficiency and better accuracy of membership details. The majority of communication within the committee is now by e-mail, and we have a website, **www.SPUMS.org.au**, which will be the central point for information for members and others. This year for this meeting, as you have seen, we now have advanced projection technology to go with our public address system which allows recording of all words spoken into a microphone.

The future

SPUMS has grown to the size of needing a central administrator, as the President has already told you. This will facilitate efficiency and accuracy. It will streamline all the membership processing and dealing with enquiries. We hope that this will allow the other office holders to concentrate on their actual official roles.

The last six years as Secretary of SPUMS has been very challenging and rewarding. I look forward to the future, to the administrator starting work and to leaving the day to day chores to him. Also for the chance to concentrate on specific issues and enhancing the image of SPUMS in cyberspace.

Cathy Meehan
Secretary of SPUMS

SPUMS

ANNUAL SCIENTIFIC MEETING 2000

will be held at

Castaway Island, Fiji from May 6th to 13th 2000

Guest speaker Professor David Elliott

Convenors are Drs Vanessa Haller and Guy Williams. Members wishing to present papers should contact Dr Haller at 55 Two Bays Crescent, Mount Martha, Victoria 3934.

The travel agent is Allways Dive Expeditions.
168 High Street
Ashburton, Victoria 3147, Australia
Tel +61-(0)3-9885-8863
Toll Free 1800-338-239
Fax +61-(0)3-9885-1164
E-mail allways@netlink.com.au

INTRODUCTORY COURSE IN DIVING AND HYPERBARIC MEDICINE

Department of Diving and Hyperbaric Medicine
Prince of Wales Hospital
Barker Street, Randwick NSW 2031
Monday 21st of February to Friday 3rd of March 2000

Objectives of the course

To provide a broad introduction to the theory and practice of diving and hyperbaric medicine (DHM)
To provide the formal teaching component required for the SPUMS Diploma of DHM
To promote integrated teaching of DHM
To promote the evidence-based practice of DHM

Course content includes

History and chamber types
Physics and physiology of compression
Decompression illness
Assessment of fitness to dive
Other accepted indications for hyperbaric oxygen (HBO) therapy
Wound assessment including transcutaneous oximetry
Practical sessions including in chamber treatment

Cost **$A 1,500.00**

For further information contact
Miss Gabrielle Janik
Phone +61-2-9382 3880 Fax +61-2-9382-3882
E-mail janikg@sesahs.nsw.gov.au

DIVING MEDICAL CENTRE

SCUBA DIVING MEDICAL EXAMINER COURSE

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

Bond University
Gold Coast, Queensland.
Easter weekend 2000.

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

Information and application forms for courses can be obtained from

Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road
Jindalee, Queensland 4047
Telephone (07) 3376 1056
Fax (07) 3376 4171

"FIT TO DIVE"

A 2-day meeting on
MEDICAL ASSESSMENT OF FITNESS TO DIVE
is being arranged by Biomedical Seminars
in association with The Medical Subcommittee of the
European Diving Technology Committee
at
The Royal Society of Medicine, London
8th & 9th April, 2000

In 2000, the annual "Fit to Dive" meeting will be held at the Royal Society of Medicine. With the subsequent retirement of the organisers, Nick McIver and David Elliott, this may be the last in this series that has survived 20 years and so we are planning it to be the best yet.

Five years have passed since the Biomedical Seminars meeting "Medical Assessment of Fitness to Dive" which was sponsored by the HSE in Edinburgh. It was a significant step towards new medical guidance on fitness assessment which was then issued to all HSE Approved medical examiners of divers. The new Diving Regulations in the UK have ensured that these annual medical assessments are applied to the wide range of working divers, from those in the offshore oil and gas industry to diving scientists and the professional instructors of recreational divers.

The Edinburgh meeting recognised that there was no great place for pass/fail criteria but that each diver needed individual assessment related to their work and that, to achieve this, the judgement of the medical examiner is paramount. Since then, training objectives for diving doctors have been approved by the European Diving Technology Committee. This 2-day meeting is for all medical examiners of divers including those Approved in the UK by the HSE. It will focus on areas of continuing controversy, such as late onset diabetes in relation to the established but inevitably ageing diver. The resumption of diving after illness, injury, surgery or a diving-related incident will be highlighted as perhaps the examiner's most challenging assessment. The medical subcommittee of the European Diving Technology Committee hopes that the output of this meeting will lead to a greater international harmonisation of standards.

The academic program will start at 0900 on Saturday 8th and end at 1700 on Sunday 9th April, 2000. The Registration fee of £180 (if paid before 31 December) will include lunch on both days. From 1 January 2000 registration will be £210.

Further details from

BIOMEDICAL SEMINARS, 7 Lyncroft Gardens, Ewell, Surrey, KT17 1UR, England.
Telephone (+44) 181 393 3318 : Fax (+44) 181 786 7036
E-mail: Karen@biomedseminars.demon.co.uk

BOOK REVIEWS

VENOMOUS CREATURES OF AUSTRALIA. 5th Edition.
Straun Sutherland and John Sutherland.
ISBN 0 19 550846 7. 1999.
Oxford University Press, Melbourne.
131 pp. 64 colour plates. RRP $Aust 24.95.

Venomous Creatures of Australia is a small paperback, easily fitting in a car glove box or jacket pocket. This guide is definitely a reference to take whenever you are out in the bush or ocean, it is a useful and compact reference tool. However, if you choose to read it before departure you may choose not to venture out at all!

First published in 1981 it has been printed in revised editions in 1982, 1985 (reprinted in 1989) and 1994, all produced by Professor Struan Sutherland. In this edition he has been joined by his son John. Obviously a book in steady demand. The subtitle A Field Guide with Notes on First Aid exactly expresses the book's scope.

The book is divided into a number of sections, the first being on First Aid Management of Bites and Stings and provides some very useful basic management advice, including The Pressure-Immobilisation Methods, when to use and when not to use. The next section relates to notes for medical and paramedical staff, and provides more specific advice on management and some useful contact telephone numbers. The guide then covers the different types of venomous creatures, beginning with snakes, ants, bees, wasps, spiders, ticks, miscellaneous land creatures, then jelly fish, octopuses, stinging fish and other sea creatures.

The book is a useful source of basic information on the 64 venomous creatures reviewed. Each entry has a description of the creature, its photograph, its habitat, with a map of its distribution, and other useful information. For each animal there is, at the foot of the last page of the entry, advice on the first aid for the bites/stings. This advice refers the reader to the appropriate page in the First Aid Management section. A syringe logo marks those animals where an antivenom is available.

This book should be in the home, car or boat. The statement in the last paragraph of the preface "..the reader who is careful is almost guaranteed immunity from bites and stings" is certainly true for the venomous creatures in this book.

Guy Williams

Key Words
Book review envenomation, first aid, land animals, marine animals.

20,000 JOBS UNDER THE SEA: A HISTORY OF DIVING AND UNDERWATER ENGINEERING.
Torrance R Parker.
Sub-Sea Archives, PO Box 2471, Rancho Palos Verdes, California 92075-6298.
Price from the Publisher $US 87.00 plus postage and packing ($US 20.00 overseas).

This is a magnificent book, a little bit bigger than A4 and 30 mm deep. It is expensive, but it is excellent value for money and the order form on the flyer ends with "I understand that I may return my order for a full refund - for any reason, no questions asked". The author, whose company published the book, is most unlikely to be called upon to refund a purchaser.

Written by a diver who started diving for a living at the tender age of 16 with the Greek sponge divers of Florida, this book leads the reader rapidly through the ages to the introduction of compressed air and the diving helmet and suit, then a short history of diving to the present day. Part 1 of the book is The Hand Pump Era, when the diver's safety at depth depended on the strength and endurance of two or more men working the handles of a pump. The deeper the diver the harder they had to work to keep up a sufficient air supply to prevent carbon dioxide build up. Muscle power could not be relied upon to properly serve the hard working diver much deeper than around 100 ft (30 m). It was the petrol engine that allowed divers to work at greater depths and face greater dangers. Hand pumps were being replaced before the end of World War I although small outfits, and most US commercial divers in those days were self employed, continued to use hand pumps for jobs away from base until World War II.

Most of the book is about work underwater. Until the 1960s most work underwater was with bridge footings and harbour construction. A side effect of harbour construction was the need for sewage outfalls to serve the growing population and then ocean intakes and outfalls to provide cooling water for power stations. The problems and dangers of placing large pipes into position for up to a mile or more on the sea bed and the solutions found by divers take up a large part, a fascinating part for those with mechanical inclination, of the book. Of course there are accidents recorded. Also recorded is the progress made by pioneers in the treatment of decompression accidents and the slow process of improving safety at work. In the 1960s oil exploration moved out to sea in a big way. This created many new jobs for divers and thousands of new divers were needed.

The fact that the book is mostly about the diving construction industry in California gives no narrow focus. Because of the small size of the commercial diving

industry in the USA divers, travelled and turned their hands to everything underwater, salvage, building pipeline and docks, inspecting dam outlets etc. Of interest is the way that unionism spread through the diving industry during the depression and New Deal years of the 1930s. Apparently new laws gave unions new powers and, as the dock construction industry had been unionised since the early 1920s, the divers had to join a union, the Pile Drivers, who were later taken over by the United Brotherhood of Carpenters. There were benefits in the form of fixed wages and better conditions of work but difficulties in getting a union ticket.

Torrance Parker considers that the period after World War II to the present, where divers are being replaced in much oil field work by remotely operated vehicles, as "Diving's Golden Age". He, among many others mentioned in the book, have made large contributions to the advancement of safety in diving by introducing new equipment, techniques and procedures.

Surprisingly those who did construction work wearing hard hats (standard diving dress) with motorised air pumps found that, in spite of the weight out of the water of all the lead needed to achieve submersion, they were able, by skilful manipulation (if that is the right word for using one's head to push a button) of inlet and outlet valves it was possible to achieve perfect buoyancy.

The book discusses two groups of hard hat divers who have disappeared. The Greek sponge divers of Florida came in the early years of the century when sponge diving was having to go deeper in the Mediterranean and the Florida sponge beds had just been discovered. The Florida sponge beds were destroyed in the late 1940s by a blight and the industry vanished. The abalone divers in Southern California were mostly Japanese who worked from small boats in teams with two or three divers who all used the same suit one after the other. Now it is illegal to dive commercially for abalone in Southern California.

In the 1960s, with the advent of deep offshore diving, some ex-abalone divers using helium recirculating helmets with carbon dioxide absorbers, based on US Navy helmets, designed modified helmets which were quieter and worked more effectively than the originals.

This brief review has just touched on a few of the many, many fascinating facts to be found between the covers. Everyone who has an interest in how working divers and water have interacted to produce the world we enjoy living in, should buy this book. Not only is it interesting, it is well written, well illustrated (nearly 400 illustrations) and has a comprehensive index.

John Knight

Key Words

Book review, equipment, diving operations, history, occupational diving.

SEA SNAKES

Harold Heatwole
ISBN 0-86840-776-3. 1999.
UNSW Press, University of New South Wales, Sydney, New South Wales 2052, Australia.
167 pp, 12 colour plates. RRP $Aust 29.95

This is one of a series of 10 books published by the University of New South Wales Press on Australian Natural History. The Series Editor, Professor Terence Dawson, stated that the ".. function of this series of titles is to make accessible accurate scientific information, complemented by high quality illustrations, on a wide variety of Australian animals. ... they are intended for students and biologists at both secondary and tertiary levels and, in general, for readers with a serious interest in animals and the environment." This book adequately achieves that goal.

Well presented and illustrated, it is easily understood by the non-marine biologist (have you ever noticed how marine biologists like using names that no one recognises and just confuses the issues?). The medical management of bites is briefly covered but then this should be expected. However, the first aid section was poorly done and lacked suitable illustrations. I believe that it is important that any book on Australian venomous animals should have a detailed description of the first aid management of injuries from that particular animal.

For all divers, Chapter 8: "Diving Adaptations", is a 'must read'. It outlines how the sea snakes avoid getting decompression illness (or "The Bends" as Professor Heatwole calls it). This chapter also highlights the difference between aquatic diving reptiles and mammals. The description of how the snakes control their buoyancy by varying their breathing pattern and lung volumes should be an example for all divers. (Sea snakes do not rely on any buoyancy aid!)

I recommend this book to all divers and non-marine biologists. It will disperse any fear about being attacked by snakes while swimming near them and perhaps will make divers realise what magnificent creatures they are. For the readers who want to know more about the biology and physiology of sea snakes without being bothered by long unpronounceable names then this book is for you. My only other criticism of the book is that it is a soft back publication which may not withstand constant handling over the years.

Chris Acott

Key Words

Book review, marine animals.

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CEREBRAL PROTECTION BY LIGNOCAINE DURING CARDIAC OPERATIONS

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Abstract

Background. Lignocaine improves outcome in animal brain injury models. Cardiac operations often cause post-operative neuro-psychological (NP) impairment. We investigated cerebral protection by lignocaine in cardiac surgical patients.

Methods. Sixty-five patients undergoing left heart valve procedures completed 11 pre-operative NP tests, a self-rating inventory for memory, and inventories measuring depression and anxiety. These were repeated 10 days, 10 weeks, and 6 months post-operatively. Patients received a 48-hour double-blinded infusion of either lignocaine in a standard antiarrhythmic dose or placebo, beginning at the induction of anaesthesia. A post-operative deficit in any test was defined as a decline by more than or equal to the group pre-operative standard deviation. In addition, sequential post-operative percentage change scores were calculated for each patient in all NP tests and the inventories for memory, depression and anxiety.

Results. Forty-two patients completed all three reviews, 8 completed two reviews, and 5 patients were reviewed once. Significantly more placebo patients had a deficit in at least one NP test at 10 days ($p < 0.025$) and 10 weeks ($p < 0.05$). The lignocaine group achieved superior sequential percentage change scores in 6 of the 11 NP tests ($p < 0.05$) and in the memory inventory ($p < 0.025$). There were no group differences in the remaining NP tests or the depression and anxiety inventories.

Conclusions. These data demonstrate that cerebral protection by lignocaine, unrelated to any effect on depression or anxiety, at a level that is noticed by the patients.

Key Words

Bubbles, drugs, research, treatment sequelae.

Introduction

Neurological problems, such as delirium, cognitive difficulty, convulsions, persistent somnolence and stroke, were reported more than 30 years ago after cardiac procedures.[1] Despite efforts to prevent sequelae, post-operative stroke and cognitive dysfunction are still seen in up to 4.9%[2] and 59%[3] of patients, respectively. Although there are some data to the contrary[4] patients undergoing cardiac operations exhibit more stroke syndromes,[5] new clinical neurological signs,[6] and neuropsychological deficits[6] than non-cardiac surgical controls, particularly in the early post-operative period. Cerebral embolism and hypoperfusion are the most likely explanations for this difference[7] and peri-operative cerebral emboli exposure correlates with the risk of post-operative stroke[2] and cognitive deficit.[8] Strategies to prevent embolic brain injury in cardiac operations have included intra-operative hypothermia;[9] filtration of the cardiopulmonary bypass (CPB) arterial line;[10] reduced manipulation of the atheromatous aorta;[11] improved removal of residual air and debris from the heart after open chamber procedures;[12] carbon dioxide field flooding during open chamber procedures;[13] and prevention of bubble formation in CPB machines.[14]

There is also interest in pharmacological cerebral protection in cardiac operations. Thiopentone reduced the incidence of early post-operative neuropsychiatric problems,[15] although patients were slower to wake, remained intubated longer and required more inotropic support than controls. This result was not replicated in a subsequent trial and routine use of thiopentone for this purpose is not recommended.[16] Nimodipine produced equivocal preservation of memory function 6 months post-operatively in a small controlled cardiac surgical trial.[17] However, a larger placebo-controlled trial of nimodipine in this context was terminated early because of higher rates of death and major bleeding in the treatment group.[18]

Lignocaine, used as a local anaesthetic and class Ib antiarrhythmic agent, has been shown in vivo to preserve neuroelectric function;[19] reduce infarct size;[20] preserve cerebral blood flow;[21] reduce cerebral oedema;[22] and reduce intracranial pressure[21] in models of cerebral arterial gas embolism,[19] focal,[20] and global[21] brain ischaemia and brain oedema.[22] Possible mechanisms for cerebral protection by lignocaine include deceleration of ischaemic trans-membrane ion shifts;[23] reduction in cerebral metabolic rate;[24] modulation of leucocyte activity;[25] and reduction of ischaemic excitotoxin release.[26] There are reports of the successful use of lignocaine as an adjunct to recompression in divers with neurologic decompression illness[27] and lignocaine has received speculative mention as a possible cerebral protective agent in cardiac operations.[28] However, there are no controlled clinical data to support these uses of lignocaine. This is a report of a randomised, prospective, double-blind trial of lignocaine versus placebo in cerebral outcome after left heart valve operations.

Material and Methods

PATIENTS

Sixty-five patients scheduled for left heart valve operation gave written informed consent for participation in the study, which received ethics committee approval in August 1994. The exclusion criteria were as follows: age outside the 20- to 70-year range; any current neurological disorder; a first or most commonly used language other than English; residence outside the greater Auckland area; and any past history of adverse reactions to lignocaine.

NEUROPSYCHOLOGICAL TESTING

All consenting patients underwent pre-operative neuropsychological (NP) testing on the day before operation. The test battery was selected on the basis of demonstrated efficacy in similar subject populations and negligible training effect and is listed in Table 1 (p 216). Six "performance" tests[29] with 11 sub-scales were chosen to measure cognitive function. A self-rating inventory with two sub-scales for memory function[30] was chosen to identify changes that were noticed by the patients themselves. Any spouses were also asked to rate the patient's memory using the latter inventory. Two inventories, one for depression and one for anxiety (two sub-scales)[29] were also used because both states influence NP test performance. All tests were repeated at 10 days, 10 weeks, and 6 months after operation, except the memory inventory, which was only repeated at 10 weeks and 6 months. Parallel forms of the Rey Auditory Verbal Learning Task[29] and Rey Figure test[29] were used in sequential testing to minimise any practice effect. Where possible, testing in the hospital was conducted in the same office. Some of the 10-day tests, and all of the 10-week and 6-month tests were performed in the patients' homes. A functional decrement was considered to exist in any of the post-operative performance tests if the patient scored at least one standard deviation (of the pre-operative population mean for that test) below their pre-operative score.[31] In addition, each patient's preoperative scores were normalised to 100 and subsequent scores were recorded as percentage changes. All NP testing was conducted by the one psychologist.

TRIAL MEDICATION ADMINISTRATION

The medication was repackaged by a pharmaceutical laboratory into coded vials. Dextrose 5% was used as the placebo solution to replicate the same mixing phenomenon as is seen when lignocaine is diluted in 0.9% sodium chloride solution. Patients were block randomised by surgeon to receive lignocaine or placebo, so that each of the five surgeons involved operated on the same number of patients in each group. The trial infusion was begun at induction of anaesthesia and continued for 48 hours. The infusion protocol was designed to deliver a 1 mg/kg "bolus" over 5 minutes, followed by 240 mg over the first hour and 120 mg over the second hour, and then 60 mg/h thereafter if the patient was receiving lignocaine. The target plasma concentration (6 to 12 μmol/L) was selected on the basis of successful in vivo[19,20] and in vitro[23] trials of lignocaine in brain injury. Blood specimens for lignocaine assay were taken to coincide with aortic cannulation and aortic declamping and at both 8 and 24 hours after starting the infusion. The latter two results were used to adjust the infusion rate. To preserve double blinding, the laboratory also reported sham levels for placebo patients.

ANAESTHESIA AND OPERATION

Patients were premedicated with a benzodiazepine (usually midazolam), an H_2 receptor antagonist (usually famotidine), and in most cases, droperidol. Anaesthesia in all patients was based on moderate doses of fentanyl (10 to 50/μg/kg) and a non-depolarising muscle relaxant, supplemented when necessary with isoflurane and benzodiazepines. Any departure from this standard protocol was recorded. The CPB circuit included a hard shell combined venous and cardiotomy reservoir (Medtronic Blood Systems, Anaheim, California), roller pump (Stockert Instrumente, Munich, Germany), hollow fibre membrane oxygenator (Medtronic Blood Systems), and a Bentley AF1040D 40 micron screen arterial filter (Baxter Healthcare Corporation, Irvine, California) with a continuous purge. Perfusion was non-pulsatile with indexed flows set at 2.4 l/m^2/min during cooling and rewarming, and 2.0 l/m^2/min during stable CPB. The alpha-stat pH management protocol was used for all patients. All patients underwent hypothermic CPB. The lowest temperature was recorded.

A Flowlink 300 colour flow Doppler machine (Rimed, Tel Aviv, Israel), operated in the 2-MHZ pulsed wave mode and interfaced to a purpose built emboli signal counter,[14] was used to monitor the right common carotid artery from 5 minutes before cannulation of the great vessels until 20 minutes after weaning from CPB. Physiological parameters were recorded during surgery by automatic data logging devices (HP Component Monitoring System, Hewlett Packard, Andover, Massachusetts). The product of time (minutes) during which perfusion pressure was below 50 mm Hg and the degree of hypotension (difference between 50 mm Hg and the observed perfusion pressure) during CPB was calculated.[32] This product is expressed as mm Hg minutes (mm Hg.min) and is known as the TM^{-50}. The cumulative duration of hypotension (systolic BP less than 80 mm Hg) before and after CPB was also calculated.

STATISTICAL ANALYSIS

The group mean scores for each test sub-scale at the pre-operative assessment were compared using an unpaired two-tailed t test. The groups were compared with respect to potentially confounding variables using a χ^2 or Fisher's

TABLE 1

TESTS AND SUB-SCALES OF THE NP TEST BATTERY [29, 30]

Test	Sub-scales	Modality Interrogated
Performance tests		
Rey figure	Copy	Visuospatial memory
	Recall	
Inspection time	Traditional	Information processing speed
	Dynamic	
Rey Auditory Verbal Learning Task	Trials 1-5	Verbal learning
(AVLT)	Distract list	Verbal memory
	Recall trial	
Symbol DigitModality Test	Oral	Complex scanning and visual tracking, manual agility
(SDMT)	Written	
	Written	
Trails A	Nil	Attention and spatial perception
Trails B	Nil	Sustained attention, spatial perception, visuomotor tracking
Self-rating inventory		
Memory Assessment Clinic	How good at?	Memory
Self Rating Test (MAC-S)	How often do?	
Control tests		
Beck depression	Nil	Depression
State Trait Anxiety Inventory (STAI)	State	State anxiety
	Trait	Trait anxiety

exact test for proportions and an unpaired two-tailed t test or a Mann Whitney U test for continuous variables. Any pre-operative or surgical factor that differed significantly between the lignocaine and placebo groups was tested by univariate regression analysis (continuous variables) or by appropriate stratification (categorical variables) against outcome for each test and at all testing times. Factors showing a significant correlation or association ($p < 0.1$) with outcome, independent of lignocaine administration, were then tested by multivariate analysis.

Analysis of the NP test outcome data was approached in two ways. First, the proportion of patients in each group exhibiting a decrement in at least one or in at least two performance test sub-scales were compared at each review using a χ^2 test. Second, in each of the performance tests, control tests and memory self-rating inventories, the sequential group mean percentage change scores were compared using repeated measures analysis of variance. This analysis was also used to assess any effect of time after operation on performance. Where a patient missed one of the three reviews, missing data were estimated from the average of the two that were completed and a degree of freedom was subtracted in the analysis of variance.[33] Patients missing two of the reviews were excluded from this analysis. A significance level of p less than 0.05 was chosen for all tests.

Results

Ten of the 65 consented patients did not enter the review phase of the trial. One withdrew after pre-operative NP testing and did not receive the trial infusion. Five of the remainder received the placebo and 4 received lignocaine. One placebo patient was unblinded in theatre after an episode of ventricular fibrillation before CPB. Two patients died after sudden cardiac arrest in the early post-operative period; one, who died on day 2, was receiving the placebo and the other, who died after discharge on day 7, had received lignocaine. Two patients had severe non-cerebral post-operative complications that would have significantly altered NP performance, 2 refused all post-operative testing, and 2 patients were lost to follow-up.

The remaining 55 patients completed pre-operative NP testing and the trial infusion (28 received lignocaine and 27 received the placebo). Forty-two patients completed all three post-operative reviews, 8 were reviewed twice, and 5 patients were reviewed once. This represents 147 of 165 possible patient reviews (89.1%). Failure to complete the review program was variously attributable to difficulty in locating patients, refusal to undergo testing, and development of non-cerebral post-operative complications.

Group mean pre-operative NP test scores for these 55 patients did not differ and are listed in Table 2. Other

relevant demographic, operative, and post-operative data are listed in Tables 3, 4, and 5, respectively. The placebo patients had a significantly greater body mass index than the lignocaine patients (28.5 versus 25.3). Conversely, myocardial scores[34] indicated significantly worse coronary artery disease in the lignocaine patients. A significantly greater proportion of lignocaine patients underwent concomitant valve replacement and coronary grafting procedures. The lignocaine group patients had a significantly longer mean duration of aortic crossclamping. A significant (inverse) correlation with outcome was shown for only one factor, in one test, and at one testing time after controlling for lignocaine administration (body mass index in the MAC-S How Good self-rating test at 10 weeks, $p = 0.014$).

There were no other significant differences in demographic or surgical variables. In particular, the TM-50, total operative emboli exposure, and the use of other putative brain-protecting anaesthetic agents, such as ketamine, etomidate and propofol, did not differ between the groups. The lignocaine patients spent a significantly shorter immediate post-operative period in the intensive care unit. There were no other significant differences between the groups with respect to post-operative variables.

Mean plasma lignocaine levels (micromoles per litre) in the lignocaine patients were 16.6 (8.5 standard deviation), 9.4 (3.3 SD), 7.8 (3.0 SD) and 10.6 (2.6 SD) at aortic cannulation, aortic declamping, 8 hours and 24 hours after initiation of the infusion.

One female placebo patient was recorded as suffering a mild peri-operative stroke, which resulted in new unilateral sensory changes. The number and proportion of lignocaine and placebo patients exhibiting a decrement in at least one or at least two performance test sub-scales at each review are presented in Table 6. A smaller proportion of the lignocaine group exhibited decrements by either definition at all times. This was significant for decrements in at least one sub-scale at 10 days ($p < 0.025$) and 10 weeks ($p < 0.05$).

The sequential group mean percentage change scores in the NP "performance" tests are either shown in Figure 1 (five of the six sub-scales in which group differences were significant) or are listed in Table 7 (the five sub-scales in which differences did not reach significance). The sixth sub-scale, in which the groups did differ significantly ($p < 0.05$), was the Trials 1 to 5 component of the Rey Auditory Verbal Learning Task, which cannot be graphed easily. In all tests where group differences were significant, the lignocaine patients' performance was superior. A significant time-dependent improvement in function was recorded in: Inspection Time (traditional); Trails A; Trails B; Auditory Verbal Learning Task (distract

TABLE 2
COMPARISON OF GOUUP MEAN RAW SCORES FOR ALL TEST SUB-SCALES IN LIGNOCAINE AND PLACEBO GROUPS AT THE PREOPERATIVE ASSESSMENT[a]

Test	Units	Lignocaine Group	Placebo Group
Performance tests			
Auditory Verbal Learning Task (trials 1-5 total)[29]	Number correct	39.4 ±9.3	40.4 ± 8.5
Auditory Verbal Learning Task (distract list)	Number correct	4.1 ± 1.3	4.6 ± 1.9
Auditory Verbal Learning Task (recall trial)	Number correct	8.0 ± 3.3	7.8 ± 2.6
Inspection time (dynamic)	Time (ms)	83.5 ± 27.1	82.9 ± 24.3
Inspection time (traditional)	Time (ms)	88.4 ±47.3	102.8 ± 51.6
Rey figure (copy)[29]	Score	32.8 ± 2.7	32.3 ± 4.1
Rey figure (recall)	Score	17.4 ± 5.7	16.0 ± 6.5
Symbol Digit Modality Test (oral)[29]	Number correct	48.7 ± 11.0	49.2 ±11.9
Symbol Digit Modality Test (written)	Number correct	41.0 ± 11.9	41.3 ± 11.7
Trails A[29]	Time (s)	31.6 ± 13.6	34.2 ± 11.5
Trails B	Time (s)	112.8 ± 102.5	103.5 ± 69.7
Self-rating inventory			
MAC-S (How good at?)[30]	Score	2.49 ± 0.58	2.42 ± 0.56
MAC-S (How often do?)	Score	2.61 ± 0.52	2.50 ± 0.47
Control tests			
Beck depression[29]	Score	7.2 ± 4.7	7.6 ± 6.8
State Trait Anxiety Inventory (state anxiety)[29]	Score	38.7 ± 11.4	38.4 ± 13.9
State Trait Anxiety Inventory(trait anxiety)	Score	35.3 ± 8.3	37.1 ± 8.3

a Data are means ± standard deviation. There were no significant differences.
MAC-S = Memory Assessment Clinics Self-Rating Test.

TABLE 3
COMPARISON OF LIGNOCAINE AND PLACEBO GROUPS WITH RESPECT TO DEMOGRAPHIC AND PRE-OPERATIVE VARIABLES[a]

Pre-operative Factor	Lignocaine Group (n - 28)	Placebo Group (n = 27)
Age (years)	56.9 ± 8.9	54.4 ± 9.7
Men	17 (60.7%)	14 (51.9%)
Women	11 (39.3%)	13 (48.1%)
Body mass index	25.3 ± 4.3[b]	28.5 ± 5.2[b]
Secondary education (years)	3.78 ± 3.0	3.81 ± 2.4
Smoking (pack-years)	4 (range 0-40)	0 (range 0-40)
Cardiothoracic ratio	53.6 ± 5.3	55.1 ± 5.4
Mean aortic gradient in patient undergoing aortic valve procedures (mm Hg)	55.1 ± 15.7	55.2 ± 14.5
Admission systolic blood pressure (mm Hg)	124 ± 16	127 +- 18
Fractional shortening (%)	33.8 -+ 10.8	37.8 +- 10.5
Atrial fibrillation	6 (21.4%)	7 (25.9%)
Renal dysfunction	3 (10.7%)	1 (3.7%)
Carotid bruit	1 (3.6%)	2 (7.4%)
Clinical left ventricular failure	6 (22.2%)	4 (14.8%)
Coronary artery disease	13 (46.4%)	8 (29.6%)
Myocardial score[34]	3 (range 0-12)[c]	1 (range 0-11)[c]
Previous transient ischemic attack	1 (3.6%)	3 (11.1%)
Diabetes	2 (7.1%)	1 (3.7%)
Peripheral vascular disease	1 (3.6%)	1 (3.7%)
Hypertension (past history)	2 (7.1%)	6 (22.2%)

[a]Data are mean ± standard deviation, number (%), or median (range); [b]p <0.025; [c]p <0.05 otherwise not significant.

TABLE 4
COMPARISON OF LIGNOCAINE AND PLACEBO GROUPS WITH RESPECT TO SURGICAL AND PERI-OPERATIVE VARIABLES[a]

Surgical Factor	Lignocaine Group (n = 28)	Placebo Group (n = 27)
Aortic valve replacement	20 (71.4%)	15 (55.6%)
Mitral valve replacement	6 (21.4%)	9 (33.3%)
Dual valve replacement	2 (7.1%)	3 (11.1%)
Valve plus coronary grafts	13 (46.4%)[b]	5 (18.5%)[b]
Redo operation	7 (25%)	4 (14.8%)
Ascending aorta atheroma	1 (3.6%)	3 (11.1%)
Duration of cardiopulmonary bypass (CBP) (min)	129.3 ± 42.6	109.5 ± 35.2
Cross-clamping time (minutes)	112.3 ± 35.5[b]	92.9 ± 27.8[b]
Emboli count	2,042 (range 247 - 6,959)	1,748 (range 216 - 11,349)
Coolest temperature (°C)	28.2 ± 2.5	28.6 + 2.0
Fractional fall in haemoglobin	0.34 ± 0.1	0.34 -+ 0.1
TM^{-50} mm Hg.minute	151 (range 15 - 1,600)	102.5 (range 0 - 590)
Pre- and post-CPB time systolic BP <80 mm Hg (minutes)	10 (range 0-97)	7.5 (range 2-78)
lnotropes after cardiopulmonary bypass	11 (39.3%)	10 (37%)
Etomidate used in anaesthetic	10 (35.7%)	9 (33.3%)
Ketamine used in anaesthetic	4 (14.3%)	4 (14.8)
Isoflurane used in anaesthetic	17 (60.7%)	16 (59.3%)
Propofol used in anaesthetic	10 (35.7%)	12 (44.4%)

[a] Data are mean ± standard deviation, number (%), or median (range); [b] p <10.05, otherwise not significant. BP = blood pressure.

TABLE 5
COMPARISON OF LIGNOCAINE AND PLACEBO GROUPS WITH RESPECT TO POST-OPERATIVE VARIABLES[a]

Post-operative Factor	Lignocaine (n = 28)	Placebo (n = 27)
ICU ventilation (hours)	12.6 ± 5.6	12.4 ± 6.2
ICU stay (hours)	24.1 ± 7.4[b]	29.4 ± 11.1[b]
ICU inotropes required	10 (35.7%)	7 (25.9%)
Intraaortic balloon pump required	1 (3.6%)	0
Peak AST	55.9 ± 28.7	58.4 ± 18.3
Peak AST >100	1 (3.6%)	0
Renal dysfunction in first 48 hours	9 (32.1%)	5 (22.2%)
New atrial fibrillation	7 (25%)	9 (33.3%)
Hospital stay (days)	9.0 ±2.6	9.6 ± 2.8

[a] Data are mean ± standard deviation, or number (%);
[b] $p < 0.05$, otherwise not significant.
AST = aspartate aminotransferase.
ICU = intensive care unit.

list and recall trial); Symbol Digit Modality Test (SDMT) (written and oral); and Rey Figure (recall) tests (all $p < 0.01$).

The sequential group mean percentage change scores in the two sub-scales of the Memory Assessment Clinics Self Report are shown in Figure 2. The lignocaine patients reported significantly better post-operative memory and fewer memory lapses than the placebo patients. Also, assessments of patients by their spouses using these sub-scales showed the same advantage for the lignocaine group, but the differences failed to reach our chosen significance level because of the small number of patients who had spouses (n = 27). The sequential group mean percentage change scores in the Beck depression inventory and the two State Trait Anxiety Index (STAI) sub-scales are shown in Figure 3. Although there was no difference attributable to treatment, there was a significant time-dependent decrease in both depression and anxiety ($p < 0.01$).

Comment

Patients undergoing left heart valve procedures were chosen for this study because of their high risk of peri-

TABLE 6
NUMBER AND PROPORTION OF PATIENTS IN THE LIGNOCAINE AND PLACEBO GROUPS EXHIBITING A DECREMENT IN AT LEAST ONE AND AT LEAST TWO PERFORMANCE TEST SUB-SCALES AT EACH REVIEW

	10 Days			10 Weeks			6 Months		
	Lignocaine (n = 25)	Placebo (n = 24)	p Value	Lignocaine (n = 26)	Placebo (n = 24)	p Value	Lignocaine (n= 25)	Placebo (n =23)	p Value
Timing of tests[a]	9.8 ± 2.6	9.8 ±1.7	NS	10.1 ± 1.8	10.8 ± 2.4	NS	29.1 ± 2.4	29.2 ± 1.8	NS
Decrement x 1	10 (40%)	18 (75%)	<0.025	12 (46%)	18 (75%)	<0.05	7 (28%)	11 (48%)	NS
Decrement x 2	5 (20%)	10 (42%)	NS	3 (11.5%)	6 (25%)	NS	2 (8%)	4 (17%)	NS

Timing of tests units are days for the first test and weeks for the other two tests. [a]Data are mean ± standard deviation. Decrement x 1 = Decrement in at least 1 scale. Decrement x 2 = Decrement in at least 2 scales. NS = not significant.

TABLE 7
SEQUENTIAL GROUP MEAN PERCENTAGE CHANGE SCORES FOR LIGNOCAINE AND PLACEBO GROUPS IN PERFORMANCE TEST SUB-SCALES WHERE THERE WAS NO SIGNIFICANT DIFFERENCE BETWEEN THE GROUPS[a]

	10 Days		10 Weeks		6 Months	
Test	**Lignocaine**	**Placebo**	**Lignocaine**	**Placebo**	**Lignocaine**	**Placebo**
Rey figure (copy)	100.6 ± 1.7	99.7 ± 1.8	102.7 ± 2.1	100.6 ± 2.0	101.8 ± 1.5	102.5 ± 2.0
Rey figure (recall)	102.2 ± 5.8	111.9 ± 12.2	126.6 ± 7.0	123.6 ± 7.5	134.3 ± 8.3	139.9 ± 11.0
Inspection time (traditional)	104.8 ± 7.4	118.8 ± 14.7	120.1 ± 6.2	121.3 ±10.9	124.9 ± 5.7	130.5 ± 16.2
Trails A	104.8 ± 3.4	112.1 ± 5.6	111.3 ± 5.5	112.2 ± 4.6	119.9 ± 5.8	115.8 ± 6.5
Auditory verbal learning task: recall trial	99.4 ±6.4	85.5 ± 6.8	111.0 ± 9.2	98.5 ±7.0	127.6 ± 9.4	114.1 ± 7.8

[a] Data are means ± standard error.

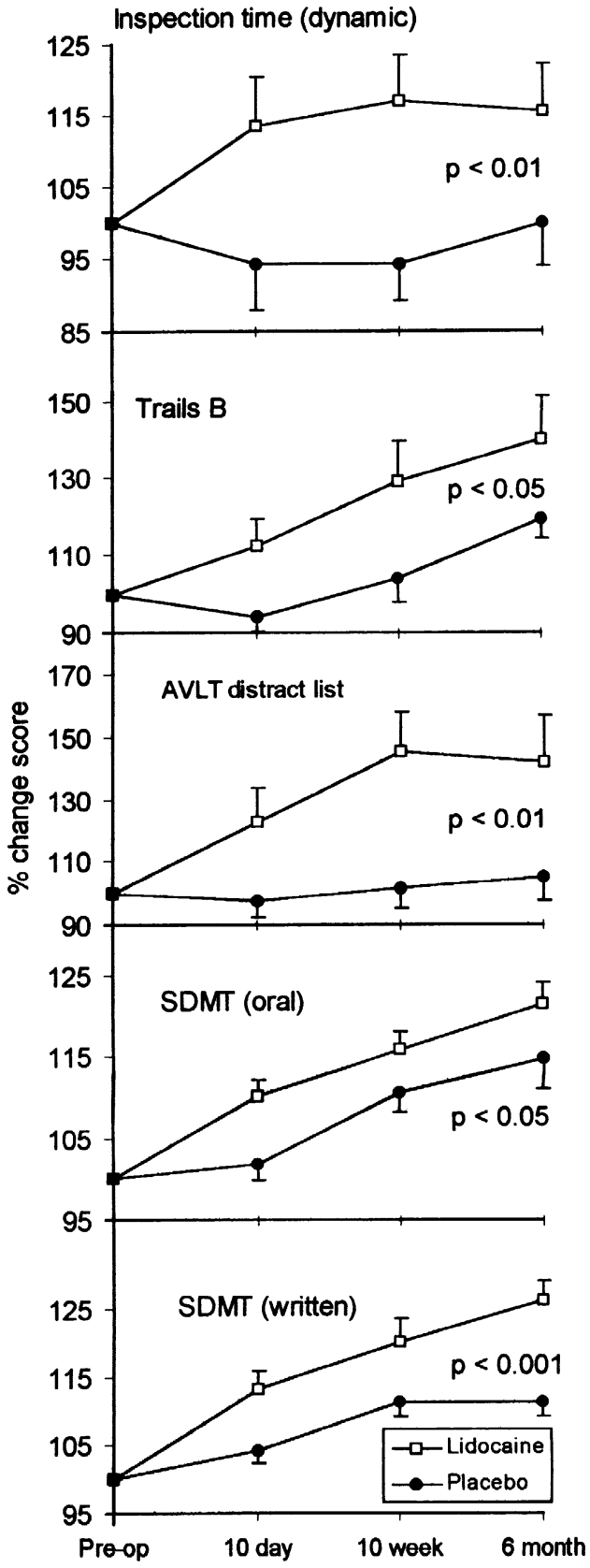

Figure 1. Sequential group mean percentage change scores for lignocaine and placebo groups in performance of test sub-scales where there was a significant difference between the groups. Data are mean ± standard error. (AVLT = Auditory Verbal Learning Task; SDMT = Symbol Digit Modality Test.)

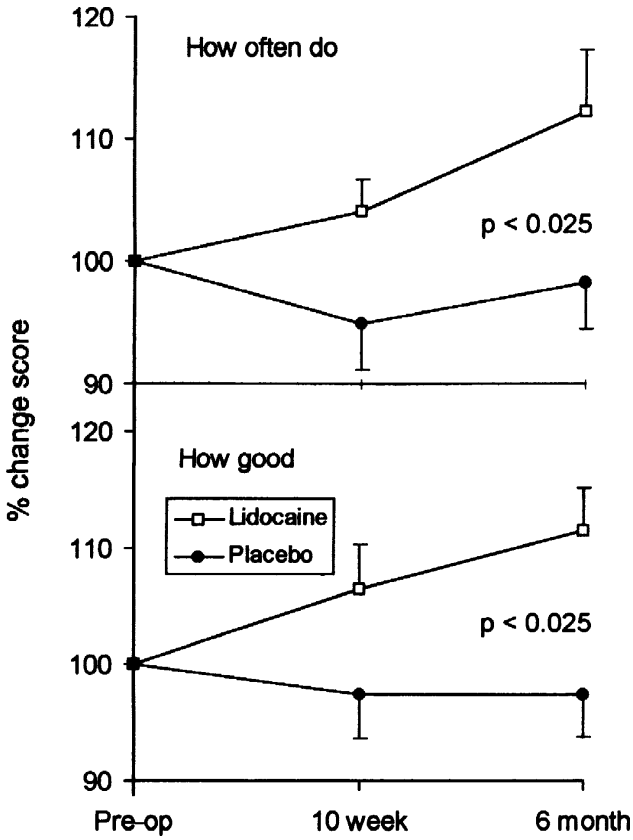

Figure 2. Sequential group mean percentage change scores for lignocaine and placebo groups in the two sub-scales of the Memory Assessment Clinics Self Report Inventory. Data are mean ± standard error.

operative brain injury.[35] In addition, the elective nature of the operation enabled pre-operative NP testing such that the patients could act as their own controls. Together, these allowed a trial of many fewer subjects than if stroke had been used as an end point.

A significantly greater proportion of the placebo group showed discrete decrements in NP test performance at the 10 day and 10 week reviews. In addition, the sequential group mean percentage change scores for patients receiving lignocaine showed improvement in all tests except the Rey Figure Copy in which a ceiling effect prevented significant change. In contrast, improvement in the placebo group mean was significantly less in some tests or absent in others. These findings suggest a strong and persistent cerebral protective effect for lignocaine. They also illustrate the previously described phenomenon of improvement in group mean NP test scores,[36] despite discrete decrements in some patients, after cardiac operations. Group mean score improvements are particularly noticeable in later reviews and are not surprising here given that we demonstrated a significant post-operative decrease in depression and anxiety in both groups. A practice effect, selective attrition, and physiological factors may also be important, but none of the latter have been identified.

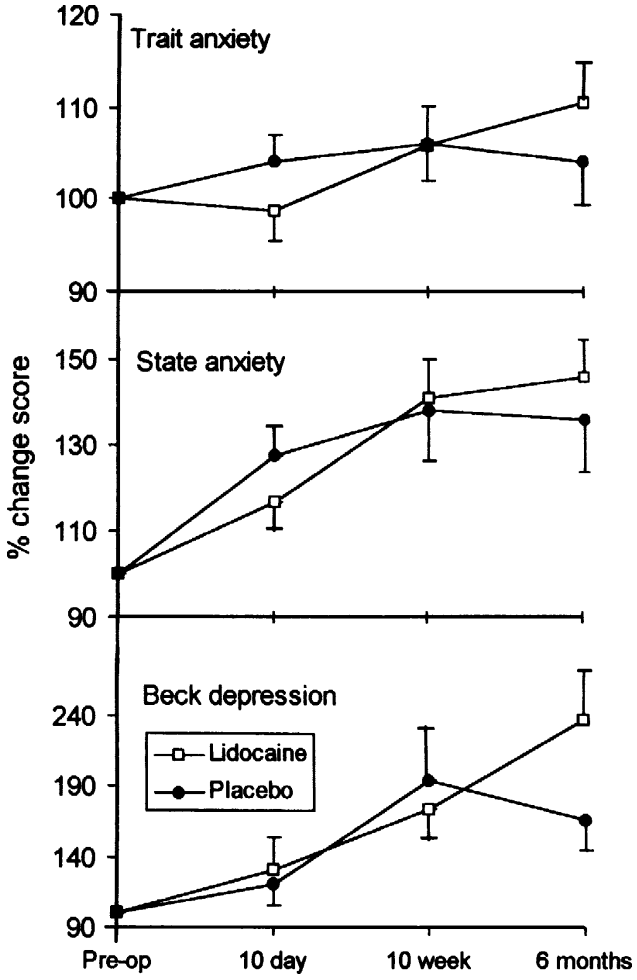

Figure 3. Sequential group mean percentage change scores for lignocaine and placebo groups in the Beck Depression and State Trait Anxiety Inventories. Data are mean ± standard error. Note that an increase in score indicates a decrease in depression or anxiety.

No confounding factors were identified that could explain the better outcome in the lignocaine patients. Although anxiety and depression may affect NP test performance,[37] the lignocaine and placebo patients did not differ either before or at any time after operation with respect to depression or anxiety indices. There is no evidence here that lignocaine directly affects mood or anxious state. Although the lignocaine patients had a significantly smaller body mass index, this has not previously been identified as a risk factor for poor cognitive outcome after cardiac operations and only a single test score (MACS-How Good, at the 10-week review) showed a significant inverse correlation with body mass index. The lignocaine patients had significantly worse coronary artery disease. This resulted in a greater proportion undergoing concomitant coronary artery grafting and valve replacement, which exposes patients to the combined risk of both procedures.[38] The lignocaine patients also experienced longer aortic cross-clamping times, which further increases relative risk of cerebral injury.[39] Although our data do not show a worsening of outcome in association with these latter factors, this is a probable consequence of subject distribution and the protective effect of lignocaine suggested by this study.

It is acknowledged that "decrements" shown to exist in NP tests may not result in any clinically discernible loss of function, such as usually seen after a stroke. However, the advantage for lignocaine shown here is not only detectable by objective NP testing, but with respect to memory at least, is also apparent to the patients themselves. In addition, a correlation between NP test results and the incidence of both objective clinical cerebral dysfunction[40] and biochemical markers of brain injury[41] has previously been demonstrated after cardiac operation.

Neither the mechanism of cerebral protection by lignocaine nor the ideal dosing regimen is demonstrated by this study. We chose a target plasma concentration consistent with that reported as effective for cerebral protection in vivo.[19,20] We arbitrarily adopted a 48-hour infusion in recognition of a possible anti-inflammatory role,[25] which might be important beyond the immediate peri-operative period. An expanded trial that will test different infusion durations is planned.

In the interim, we recommend that lignocaine be considered for the routine peri-operative care of patients undergoing left heart valve procedures. Consideration should also be given to investigating a role for lignocaine in other forms of brain injury.

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THE EFFECT OF BUBBLES ON THE LIVING BODY

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Key Words
Bubbles, decompression illness, physiology.

Introduction

There is general agreement that the basic problem in decompression is gas coming out of solution and forming a gas phase. However, it is also well known that a considerable number of bubbles can be formed without any acute signs or symptoms. Such bubbles have been called "silent" bubbles[1] and have, in particular, been observed in the pulmonary artery.[2] One conclusion that can be drawn from this observation is that acute clinical symptoms are critical dependent upon the location of the bubbles. Bubbles in the brain, for instance, could give few symptoms, as large areas of the brain are clinically silent. Bubbles in joints, on the other hand, would give symptoms, because of the rich innervation by pain receptors in these areas. One effect of this would be that we have to distinguish between primary and secondary effects of bubbles. The primary effects are related to the mechanical effect of the bubbles, which may be blockage of the circulation or distortion of tissue. The secondary effects are related to the numerous effects of the bubble surface, with activation of a large number of biochemical and cellular mechanisms. It seems obvious that this secondary effect can occur without any acute signs or symptoms.

When do bubbles form ?

Most, if not all, practical decompressions will lead to some degree of gas bubble formation in the organism

and the risk of decompression illness (DCI). The predominant theory about the growth of bubbles is that bubbles grow from preformed nuclei, as the resistance of "pure solutions" to supersaturation and gas phase development is considerable.[3] One likely theory is that the nuclei are composed of small (approximately 1 micron) stable gas bubbles.[4]

The exact threshold for bubble formation is not known, but it is probably in the range of 50-70 kPa in tissue[5] and even lower in the vascular system. Eckenhoff et al. demonstrated that saturation at 3.7 msw on air was sufficient to produce bubbles in the pulmonary artery in man.[6] The conclusion from this study must be that gas bubbles will form in the vascular system at any supersaturation and that the concept of a minimum tolerable limit of supersaturation, as least for the venous system, may only relate to clinical symptoms and not to bubble formation. Adding to this problem is the fact that it has been demonstrated repeatedly that large inter-and intra-individual differences in bubble forming "ability" exist. Factors like sex, age, body build, circulation, temperature, blood composition and degree of exercise seem to play a role.[7,8] Cavitation in joints, for example, has been demonstrated without any supersaturation following violent movements. Even under experimental conditions where the circulation is kept stable, a variation in vascular bubbles between individuals, often a factor of 10, following decompression is observed.[9] Furthermore, there are data indicating that there is a large difference is susceptibility to decompression sickness that is not directly related to the amount of vascular gas bubbles observed.[10]

Where do bubbles form ?

Bubbles have been observed in many tissues in the body following decompression. They are most commonly found in the vascular system, the white matter of the CNS, in abdominal fat, in synovial fluids and in muscles.[11] Following quite severe decompressions, we did not observe bubbles in the muscles themselves, but only on fascia.

Harvey et al. studied the limits for vascular bubble formation in cats, both at rest and after electrical stimulation and tissue injury.[12] The conclusion from these studies was that at marginal exposures, stimulation or injury was needed for bubble formation. At higher supersaturations, bubbles occurred at rest, the time of occurrence determined by the fat content. Essentially the same results have been obtained in frogs and rats.[13] Based on these studies, the authors concluded that gas bubbles are chiefly intravascular and that they are responsible for nearly all important phases of the syndrome of decompression sickness. Only in very severe cases did extravascular bubbles play a role and then only in lipid rich structures.

Venous bubbles

There is evidence from many studies that gas bubbles occur in the venous system during most decompressions.[14,15] Several studies have documented the relationship between the occurrence of many venous bubbles and the risk for clinical symptoms requiring treatment.[16,17] 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.

Arterial bubbles

Gas bubbles in the arteries have been detected in divers after excursions,[18] during decompression from saturation dives[19] and at autopsy after fatal accidents.[20] Arterial gas bubbles have also been observed in large animals during and after decompression.[21-23] Thus, there is no doubt that arterial gas bubbles occur during or after some decompressions.

In divers, there are several possible pathways by which venous bubbles may reach the arterial circulation. First, venous gas bubbles may travel through the pulmonary circulation and enter the pulmonary veins and the left atrium, although the pulmonary circulation is usually considered to be a good filter for gas bubbles as well as for other emboli. Second, venous gas bubbles may pass through a patent foramen ovale (PFO) or other extraordinary connections in the heart to reach the left side of the heart. Third, if the lung has been overinflated during a rapid ascent, gas may escape directly into the pulmonary veins after alveolar rupture.[24]

Finally, gas bubbles may form in the arterial circulation if the decompression rate is sufficiently fast >0.3-1 fsw/sec.[25] All gas nuclei in the blood will not be destroyed at compression and supersaturation of the arterial blood may occur during the rapid decompression. However, an experimental study using goats did not succeed in demonstrating such bubbles in the arterial circulation after a short hyperbaric exposure and a rapid decompression.[26]

In as many as 20-34% of humans, dependent on age, the foramen ovale is patent after foetal life.[27] Normally it is functionally closed, since the pressure in the left atrium is higher than the pressure in the right atrium and the septum primum functions as a valve. However, a spontaneous shunt, not dependent on a Valsalva manoeuvre or other factors to change the pressure gradient between the atria, is diagnosed in 5-6% of humans using contrast echocardiography.[28,29]

Tissue bubbles

It seems reasonable to assume that tissue bubbles can occur if the gas load is high enough. This was apparently shown for the spinal cord by Francis et al., who showed tissue bubbles in the white matter following rapid decompression after a 15 minute dive to 300 fsw on air.[30] This work has, however, recently been challenged by Palmer, who claims that all changes observed could be explained by gas bubbles inside vessels.[31] This is also supported by the recent work of Sharpe and Broome, who showed that there was no relationship between the fat content of the spinal cord and the occurrence of gas bubbles.[32] Even if the exact mechanism of tissue injury is controversial, the evidence seems to indicate that vascular processes are the more important ones.

Primary bubble effects

Initially, the bubbles will lead to changes, mainly due to their direct mechanical effects. When gas bubbles form and expand they can obstruct the arterial and venous circulation, leading to tissue ischaemia, or they can damage the tissue and induce pain by direct pressure effects. Bubble formation may influence circulation by mechanical obstruction. Venous obstruction may lead to oedema and arterial obstruction may lead to tissue ischaemia, both of which have been observed after decompression.[33] This can reduce gas elimination, both by increasing diffusion distances and by reducing blood flow.

One important primary effect of the bubbles, which is often forgotten, is the reduction in gas elimination caused by these bubbles. Both theoretical[34] and experimental[35] studies have demonstrated that gas bubbles in the tissue will increase gas elimination time. In another study, it was shown that bubbles significantly increased the time constant of the slow component of the bi-exponential curve describing the nitrogen concentration in the pulmonary artery.[36] This can be seen in Figure 1.

Obstruction of flow to the tissue by bubbles may increase elimination time even more. Computer simulations have shown that a high number of bubbles can increase the time constant of gas elimination from muscles from 50 to 2,000 minutes (Flook, Personal communication 1999). This is partly taken into account by the new US Navy (USN) diving tables, where gas elimination is considered to be linear, not exponential.[37] However, in reality the problem may be even more complex as bubbles in the circulation may increase the transport of gas to the lungs.[38]

Secondary bubble effects

When gas bubbles are formed protein denaturation takes place at the blood-gas interface.[39] The gas-blood

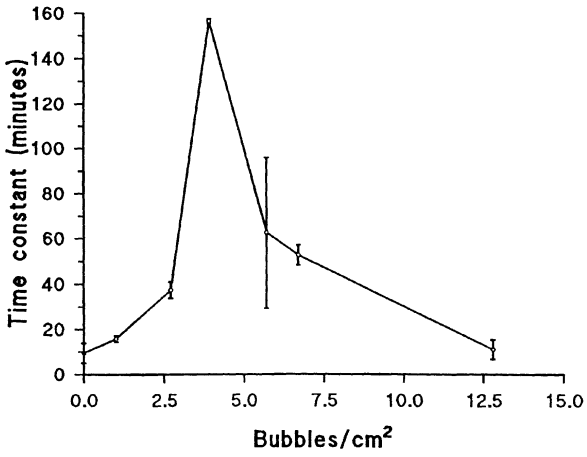

Figure 1. The relationship between the number of bubbles in the pulmonary artery and the time constant for the elimination of nitrogen from the pulmonary artery. Mean values with 95% confidence intervals.

interface is a thin layer, approximately 20 nm thick, consisting of fibrin and gamma globulin.[40,41] This layer acts as a foreign substance, activating formed elements of the blood and inducing biochemical changes such as complement activation.[42] These mechanisms play a significant role in the response of the tissue to venous gas emboli and are probably the basis for any long term effects that may occur. At present, our understanding of the importance of these mechanisms is quite fragmentary. Much further work is needed to determine the relative influence of mechanical and biochemical effects of gas embolism. An understanding of these processes is necessary for development of a rational approach to treating or preventing injury caused by vascular bubbles.

Inflammation

The response of an organism to injury is termed inflammation. In decompression sickness this inflammatory process is initiated by the surface of the bubbles, which is regarded by the organism as a foreign substance. However, this process can also be initiated by direct mechanical injury to the tissue. One important mediator of the inflammatory process is activation of the complement system. Gas bubbles activate the complement system in-vitro.[43] The degree of activation is dependent upon the amount of gas infused, varies considerably over several months in one individual and is not dependent upon the gas composition of the bubble. No relationship was seen between the degree of C5a activation in vitro and the level of C5a observed in vivo after air dives.[44]

Leucocytes are involved in many aspects of tissue injury and inflammation. Several studies have documented that leucocytes are activated by decompression. Philp et al.[45] showed that decompression led to a reduction in both

thrombocytes and leucocytes. These reductions seem to be related to the presence of gas bubbles. During decompression from a saturation dive to 440 msw, where no gas bubbles could be detected, there was no reduction in leucocyte number.[46] In another saturation dive, Benestad et al.[47] showed that decompression lead to activation of neutrophils. Contrary to what has been observed in thrombocytes, no activation of leucocytes seems to occur in vitro at pressure.

Coagulation

Aggregation of thrombocytes may lead to the formation of blood clots, thus forming solid emboli which may compound the effect of gas bubbles in the lung. Thorsen et al.[48] showed that gas bubbles lead to aggregation of thrombocytes in-vitro. Aggregation in-vitro was strongest when the bubble diameter was between 40 and 120 μm. The degree of aggregation does not seem to be dependent upon the gas content of the bubble, but only on its surface properties.[49] Aggregation of thrombocytes by gas bubbles can be considerably enhanced by adrenaline (epinephrine).[50]

Vasoconstriction/dilatation

Gas bubbles can induce vasoconstriction by direct effects on the vascular wall[51] or vasodilation or constriction by initiating the release of different vasoactive substances.[52,53]

Bubble effects on different organ systems

In the following the bubble effects will be described on some organ systems. Primary and secondary effects will be described together, as they often are quite difficult to distinguish.

Endothelial damage

Chryssanthou et al. have shown that animals exposed to decompression will show breakdown of the blood-brain barrier and the blood-lung barrier.[54] Broman et al. have demonstrated that even very short contact between gas bubbles and endothelium (1-2 minutes) will lead to such breakdown.[55] Furthermore, studies in rabbits indicate that such contact leads to endothelial damage and progressive reduction on cerebral blood flow and function. In a study by Smith et al.[56] endothelial damage could be demonstrated in pigs exposed to severe decompressions. We were able to demonstrate changes in the endothelium in pigs following exposure to gas bubbles. We found that these changes occurred at an exposure of approximately 1.5 bubbles/cm^2, equivalent to approximately Grade II-III on the Spencer scale.[57]

Even minimal endothelial injury can induce activation of both biochemical and cellular responses, which could form the basis for tissue injury following decompression.

Lung function changes

Gas emboli may block some parts of the pulmonary vascular bed, reducing or preventing blood flow through the regions of lung served by those vessels. This leads to an initial rise in the pulmonary artery pressure (PAP) and pulmonary vascular resistance (PVR), and a decrease in arterial oxygen tension (P_aO_2).[9,58,59] Following decompression, changes in diffusion capacity[60] and lung function changes similar to “small airways disease” have been seen.[61]

Central Nervous System changes.

Central nervous changes in DCI are probably caused by several mechanisms. In severe DCI, both vascular bubbles and in-vivo bubble formation probably plays a role.[33]

Exposure to vascular bubbles without clinical symptoms do not seem to have a serious effect upon the spinal cord.[62] In this group of 10 amateur and 10 professional divers, five of whom had suffered from DCI, no changes could be seen. In the brain, changes in the endothelial layer of the ventricles could be detected in a group of divers.[63] A possible explanation is that this damage is caused by gas bubbles in the spinal fluid, such bubbles will probably primarily adhere to the lining of the ventricles.

Numerous studies have shown that circulating gas bubbles change the blood-brain barrier, this is described above.

Bone

Aseptic bone necrosis is regarded as an occupational hazard for all workers under pressure.[64] There is clear indications that the incidence of bone necrosis is linked to decompression. This is perhaps best demonstrated by the fact that this disease, which is quite rare in the industrial world, is considerably more prevalent in the developing world, where diving practices produce a high incidence of decompression sickness.

Conclusions.

A recent consensus conference on long term health effects of diving[11] concluded that “changes can be seen in

lungs, CNS and bone in divers who had not had any decompression accidents", or perhaps more accurately, in divers who had not been treated for decompression illness. The mechanisms for these changes are not clear, but as bubbles in the vascular system have been observed frequently in divers, a reasonable working hypothesis would be that vascular bubbles may play a major role in this. There are still many details missing how the bubbles affect the organism. An understanding of the mechanisms involved is important, however, both for preventing injury and for treating the damage caused by these bubbles.

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37 DECOMPRESSION SICKNESS CASES TREATED IN THE DEPARTMENT OF UNDERWATER AND HYPERBARIC MEDICINE, ISTANBUL FACULTY OF MEDICINE

Akin S Toklu, Samil Aktas, Salih Aydin and Maide Çimsit

Abstract

The time interval between the onset of decompression sickness (DCS) and recompression therapy, and the first aid with medical treatments applied before reaching a recompression facility, will affect the outcome of the recompression therapy.

In this study 37 DCS cases were evaluated to find out the time interval to the onset of DCS, the type of the disease and symptoms, delay to recompression treatment, medical treatments applied during transport, recompression treatment protocols performed and outcomes.

All the patients were male. Professional divers (32 or 86.5%) outnumbered sports divers (5 or 13.5%). In 20 cases (54.1%) onset of symptoms was within the first 10 minutes after the dive. In three men (8.1%) symptoms came on underwater. Numbness, tingling and back pain were the most frequent symptoms reported by the patients with Type II DCS. Complete recovery was achieved in 32 (86.5%) of the cases by recompression therapy combined with medical treatment. Rehabilitation was needed in 12 (32.4 %) of the cases.

Omitted decompression was the most frequent cause of DCS in our cases. Additional hyperbaric oxygen therapy needed in delayed cases is evidence of the importance of immediate transport and adjunctive medical treatments.

Key Words

Decompression illness, first aid, transport, treatment.

Introduction

The hyperbaric facilities in Turkey are mostly situated in Istanbul. The only Hyperbaric and Underwater Medicine Department in civilian universities is in the Istanbul Faculty of Medicine. The Fisheries Research Institute also has a hyperbaric chamber for treating divers in Bodrum, in Aegean Sea region. The Turkish Navy has three hyperbaric units. Besides these public facilities, all with multiplace chambers, hyperbaric oxygen therapy is performed in three private hyperbaric centres, in Istanbul. The three private hyperbaric centres, which all have multiplace chambers and one also has a monoplace, are free standing and mostly use hyperbaric oxygen therapy for indications other than diving related disease. In March

Figure 1. Dive sites in Turkey.

1999 there were 11 doctors who specialised in underwater and hyperbaric medicine in Turkey. There were also four assistant doctors specialising in the subject.

Recompression treatment of the divers in this study was performed at the Department of Underwater and Hyperbaric Medicine, Istanbul Faculty of Medicine. None of the private hyperbaric facilities had been established when the patients covered in this paper were treated.

There is an inadequate decompression in most decompression sickness (DCS) cases, but DCS may also occur after a dive in which decompression is performed according to standard decompression tables. The time interval between the onset of DCS and recompression, and when first aid with medical treatments, such as 100% oxygen, fluid, steroids and acetylsalicylic acid, is applied will affect the result of recompression therapy and the course of the disease. Recompression therapy must be given for any doubtful DCS symptom, since it cannot be predicted whether the condition will deteriorate or not. In this study 37 DCS cases treated in our department were evaluated to find out the time interval of the onset of DCS, type of the disease, type of the symptoms, delay to recompression treatment, first aid and medical treatments applied during the transport, recompression treatment protocols performed, rehabilitation and outcomes.

Methods

Patient records were used for this study. The record for each patient included information about the dive site and time, dive profile, first aid and medication during transport, course of the disease, symptoms and physical findings, medical and recompression therapy, besides individual information such as age, sex and profession. We calculated the ratios concerning dive sites, type of the symptoms and onset time, delay to recompression therapy and recompression protocol performed.

Results

Although the number of female recreational scuba divers has increased in our country recently, all patients were males. The age of the divers varied between 16 and 57 years (average 31). There were 32 professional (86.5%) and 5 recreational scuba divers (13.5%) among our sample. The majority of the professionals consisted of shellfish divers mostly working on the Black Sea coast within 3 to 6 hours by an ambulance of our department. Recreational diving is carried out largely in the Mediterranean and Aegean Seas. Table 1 gives the numbers and proportions of our sample from each area and the times for overland ambulance transport. Figure 1 shows the distribution of Turkish diving sites.

There were neurological and cardiopulmonary symptoms in 18 (48.6%) of the cases. These patients were diagnosed as Type II DCS. Their symptoms are displayed in Table 2. Their most frequent symptoms were numbness, tingling and back pain. Nineteen patients had no neurological symptoms or signs (Type I DCS); of those who complained of musculoskeletal pain, the shoulder was the most often affected site.

TABLE 1

DISTRIBUTION OF CASES BY DIVE SITE

Dive Site	Cases		Road travel time to Istanbul
Black Sea	23	61%	3-14 hours
Sea of Marmara	6	17%	2-3 hours
Aegean Sea	4	11%	5-12 hours
Mediterranean	4	11%	12-14 hours
Total	37	100%	

TABLE 2

FREQUENCY OF SYMPTOMS REPORTED IN 18 TYPE II DCS CASES

Symptoms	Number	%
Back pain, numbness and tingling	6	33.3
Fatigue and muscle weakness	5	27.8
Chest pain, cough and difficulty in breathing	4	22.2
Abdominal pain and pain in extremities	3	16.7
Nausea and vomiting	2	11.1
Headache, dizziness and fainting	1	5.6

In five patients (13.5%) DCS occurred after a dive which was within US Navy (USN) no-decompression limits. But there was omitted decompression in the dive profiles of 31 divers (84%). In one case flying after diving caused DCS.

In three men (8.1%) the first DCS symptom was experienced in the water while the diver was still decompressing. Most of the cases (24 or 54.1%) DCS occurred within the first 10 minutes after the dive. One case reported the first symptom 18 hours after his dive (Table 3).

Most patients (20 or 54%) took more than 12 hours to reach to our department (Table 4).

No adjunctive medical therapy was used in 13 patients (35.1%) during the transport. Medical therapy was performed as a combination of oxygen breathing, acetylsalicylic acid, steroids and fluid therapy in only three cases (8.1%). In-water recompression on air was tried by nearly half, 15 out of 32 (40.5%), of the professional divers. Except for the diver who developed DCS during flight, land transport was used in all cases.

Initial recompression therapies are shown in Table 5. Five patients (26.3%) patients with Type I DCS required additional hyperbaric oxygen therapy. Sixteen of the 18 cases of Type II DCS (89%) required follow up treatments.

Complete recovery was achieved in 32 divers (86.5%) by recompression therapy combined with

TABLE 3

TIME TO ONSET OF SYMPTOMS

Onset of symptoms	All patients		Type I DCS		Type II DCS	
Number	%	Number	%	Number	%	
While diver is in water	3	8.1	1	5.3	2	11.1
First 10 minutes	20	54.1	9	47.3	11	61.0
10 minutes-1 hour	9	24.3	6	31.6	3	16.7
1 hour-2 hours	4	10.8	3	15.8	1	5.6
18 hours later	1	2.7		-1	5.6	
Total 37		19		18		

TABLE 4

DELAY TO RECOMPRESSION

Delay	<3 hours	3-6 hours	6-9 hours	9-12 hours	12-24 hours	24-72 hours	3-7 days	>7 days
Ratio (%)	5.4	10.8	21.6	8.1	18.9	27.1	5.4	2.7
Numbers	2	4	8	3	7	10	2	1

TABLE 5

INITIAL RECOMPRESSION THERAPY TABLES

	Type I DCS			Type II DCS	
Recompression	US Navy TT 5	US Navy TT 6	HBO Protocol	US Navy TT 6	US Navy,TT 6 with extentions
Ratio	31.6%	26.3%	42.1%	16.7%	83.3%

adjunctive medication. Rehabilitation was needed in 12 patients (32.4%). Residual symptoms such as weakness in some muscles and patches of numbness were left in 4 cases (10.8%). One case (2.7%) was resistant to the therapy and remained paraplegic.

Discussion

Omitted decompression was the main reason of DCS in our cases. When their dive profiles were compared it was clear that recreational scuba divers paid more attention to the decompression rules than professionals. Inadequate decompression increased the risk of DCS, but cases did occur after no-decompression dives, confirming that DCS can be seen even after a dive within the no-decompression limits.

The frequency of the onset of DCS symptoms in the first 10 minutes after the dive shows the importance of both the dive profile and the ascent history in the differential diagnosis of DCS and arterial gas embolism due to pulmonary barotrauma.

Most of our cases presented late for recompression therapy since the importance of quick transport is usually not realised. For the majority of the cases the time intervals between the onset of the symptoms and recompression therapy was more than 12 hours. This occurred although most cases came from a distance which only takes 3-4 hours in an ambulance. Those cases delayed by geographical factors, position of the dive sites and difficulties encountered during the transport, indicate the necessity of recompression chambers in remote areas.

The percentage of the cases who had adequate first aid with adjunctive medical treatment was very low. The poor outcome of recompression therapy, the need for additional hyperbaric oxygen therapy and rehabilitation in delayed cases are evidence of the importance of immediate transport and adjunctive medications. Appropriate first aid and medical treatment can be provided by training medical staff near the diving sites about diving related disease and its treatment. Deterioration of the cases who applied in-water recompression on air shows the harmful effect of its improper application. Air transport was not used in these cases because of its high cost and the fact that there is no well established insurance system in Turkey.

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DECOMPRESSION ILLNESS TREATED IN SOUTH THAILAND DURING 1998

Gustavo Ambriz

Key Words

Decompression illness, recreational diving, treatment.

Abstract

Decompression illness (DCI) occurred in 30 European recreational scuba divers, using compressed air, in South Thailand in 1998. This is the first report on such treatment. Of interest is the high proportion of patients who were employed in the recreational diving industry.

Introduction

Recreational diving in South Thailand is done on the west coast from live-aboard boats (predominantly near Surin, Similan and also in Burma) and from shore based dive shops. Day trips and shore diving are done off Phuket, Phan Nga Bay, the Phi Phi Islands, Hin Daeng and also in the Gulf of Thailand on the islands Ko Samui and Ko Tao. There are about 100 dive shops in the region with an estimated 1,000 dives per day are done during the high season (December-April).

The majority of recreational divers in South Thailand are foreigners visiting for a week or so.

Europeans form the majority of the dive guides and instructors. They dive regularly with longer periods between breaks than do the tourists. Inclusion criteria for the present study were decompression illness (DCI) in European recreational scuba divers breathing compressed air. Thirty such patients presented in 1998. Data about non-European nationals will be presented in a later report.

Hyperbaric Services Thailand (HST) is a private recompression facility. It has a multiplace, 1.5 m (5 ft) diameter, double-lock hyperbaric chamber in Phuket, Thailand. The nearest hyperbaric facility is in Bangkok, approximately 1,000 km away.

If one accepts the estimate of 1,000 dives a day in South Thailand in December to April there would be approximately 150,000 dives carried out in that time.

A total of 42 cases were recompressed in 1998 at HST. Of these, 41 cases were DCI and one case was peri-orbicular emphysema secondary to fracture of the floor of the orbit. Five were indigenous divers. The rest (36) were Europeans or from Canada (2), Israel (2), New Zealand (1) and the USA (1). The latter are not included in this report. All were recreational divers and are summarised in Table 1 by country and frequency. Because of the small numbers involved all percentages are given to the nearest appropriate whole number (so that the total is 100%) to avoid a spurious accuracy.

It is interesting that 12 of the 30 patients studied came from the Scandinavian countries. Britain and Germany with more than 10 times the population only provided 13 cases. There was no obvious reason for this.

TABLE 1

PATIENTS' COUNTRY OF ORIGIN

Country	Cases	%
England	7	19%
Denmark	6	17%
Germany	6	17%
Sweden	5	13%
Canada	2	5%
Israel	2	5%
Austria	1	3%
Belgium	1	3%
France	1	3%
Italy	1	3%
New Zealand	1	3%
Norway	1	3%
Switzerland	1	3%
USA	1	3%
Total	**36**	**100%**

TABLE 2

DIVER EXPERIENCE (TIME DIVING)

Experience	Cases	%
Under instruction	2	7 %
Under 6 months	7	24 %
Between 6 months and 2 years	5	17 %
Between 2 and 6 years	9	31%
More than 6 years	6	21%
Total	**29**	**100%**

The divers

Of the 29 treated divers who gave information about their experience 31% (9) had less than 6 months diving experience. Table 2 gives further details of the range of experience.

Twelve (40%) of the 30 treated European patients were locally based instructors/dive guides. Certification levels of the patients are shown in Table 3.

There were 21 males (70%) and 9 (30%) females. Age ranged from 21 to 46 years. One third of the cases (10) were between 25 and 29 years old.

Eleven of the 31 Europeans treated in the chamber (30 for DCI and one for peri-orbicular emphysema) had had previous illnesses. Table 4 gives the details. Four had had DCI on a previous occasion.

TABLE 3

CERTIFICATION LEVELS

Qualification	Cases	%
Student	2	7%
Open Water Diver	6	20%
CMAS 1 *	1	4%
CMAS 2 *	1	4%
Advanced Diver	6	20%
Rescue Diver	1	4%
Dive master	4	14%
Instructor	8	27%
Total 29	**100%**	

TABLE 4

PREVIOUS MAJOR ILLNESSES

Illness	Cases	%
Previous DCI	4	13%
Musculo/skeletal	2	7%
Asthma	2	7%
Gout	1	3%
Neurologic	1	3%
Spine	1	3%
None	20	64%
Total	**31**	**100**

TABLE 5

CURRENT HEALTH PROBLEMS

Problem	Cases	%
Fatigue	9	30%
Hangover	3	10%
Asthma	2	7%
Diarrhoea	2	7%
Neck pain	1	3%
Back pain	1	3%
None	12	40%
Total	**30**	**100%**

TABLE 6

DAYS DIVING DURING PRESENT TRIP

Number of days	Number	%
1	4	15%
2	6	22%
3	2	7%
4	2	7%
5	1	4%
Working	12	45%
Total	**27**	**100%**

TABLE 7

CHARACTERISTICS OF 28 DIVERS WITH DCI

Finding	Cases	%
Buoyancy problems	1	4%
Rapid ascent	5	18%
Single dive that day	7	25%
Within limits	27	96%
Symptoms prior to last dive	2	7%

Eighteen patients (60%) had a current health problem when they developed DCI (Table 5). Nine (30%) were suffering from fatigue.

Decompression Illness

Only 6 cases (20%) were diagnosed as DCS Type I (pain only). The majority, 24 cases (80%), had Type II DCS with neurological signs and symptoms. There were no cases of arterial gas embolism.

Only 27 divers provided information about how many days they had been diving before they developed DCI. Table 6 gives the numbers and the percentages of the 27 divers providing this information.

Nine out of 28 patients (32%) had consumed alcohol during the dive series that led to DCI.

Table 7 shows that the vast majority (96%) of the divers who provided the information were diving within the limits provided by their computers. Only one diver had buoyancy problems and 5 had made rapid ascents. Two divers had symptoms before they started the dive which led to them being treated.

Assuming that Europeans make up about the same percentage of the total as they do of those treated for DCI, the incidence of DCI in Europeans is about 0.02%. Most of the cases (27) came from Phuket, where the chamber is located, taking from 15 minutes to 3 hours to reach the chamber. The other two cases where the location was recorded came from a live-aboard based at Similan, taking 9 hours to reach the chamber, and from Kao Lak (3 hours).

There was a large variation in the time between the end of the dive and the onset of symptoms (Table 8). About a third of the patients developed symptoms within 30 minutes. Another quarter developed them in the next 3.5 hours, with a further quarter in the next 4 hours. However a tenth of the patients developed their symptoms between 16 and 24 hours after diving.

Divers delayed for long periods before reporting to the HST. Only three cases (10%) took less than two hours to reach the chamber. Only 5 patients (17%) reached the chamber within 6 hours of developing symptoms. Fifteen (50%) took two or more days to reach treatment. Table 9 gives the individual times for these delays.

Treatment

Few patients, only 9 out of 29 (31%), had had any first aid treatment during evacuation. Table 10 gives the depressing figures. There is obviously much room for

TABLE 8

TIME TO SYMPTOM ONSET AFTER SURFACING

Time	Cases	Accumulated percentage
10 min	2	8%
20 min	1	11%
First 30 min	9	35%
First 4 hours	16	61%
First 8 hours	23	89%
Between 16 and 24 hours	3	11%
Total	**26**	**100%**

TABLE 9

TIME FROM ONSET OF SYMPTOMS TO RECOMPRESSION

Minutes/cases		Hours/cases		Days/cases	
30	1	1	2	2	3
		3	1	3	6
		4	1	4	2
		6	2	5	1
		7	1	6	2
		10	1	9	1
		12	4		
		15	1		
		18	1		

TABLE 10

FIRST AID BEFORE REACHING HST

Oral fluids	3
Oxygen via free flow	3
Oxygen via demand valve	2
Aspirin	1
None	20
Total	**29**

improvement in the early management of DCI cases in the region.

All 30 patients had a US Navy (USN) Table 6 as initial treatment. For some cases the table was extended. For those who had not completely recovered USN Table 5 was used for follow up treatments. The average number of treatments was 3.83.

Seven patients had residual symptoms after recompression therapy was completed. Because these patients moved away at the end of their holiday, follow up has not been possible.

Discussion

South Thailand has a booming recreational diving industry. In 1997 we treated 22 cases of DCI, in 1998 there were 42 and the total for 1999 will be higher. More specific data collection and analysis of the factors that resulted in DCI will be undertaken. The findings of the physical examination will also be described and follow up will be included in further reports.

In 1998 divers with less than 2 years experience accounted for 21 (50%) of our injured divers. The high percentage (40%) of locally based instructors or dive guides who suffered DCI deserves a detailed analysis. In the 1998 DAN report this group accounted for only 17% of the reported cases. It is likely that the reason lies in their pattern of diving and complete reliance on their dive computers.

The long delays in seeking treatment, with less than 20% of patients presenting in the first 6 hours after developing symptoms, can be attributed more to ignorance, or denial, rather than to distance to the recompression facility. Education of the local dive providers and medical personnel is needed in prevention, symptom recognition, on site management and evacuation procedures of DCI patients.

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TEAR FILM BUBBLES AND DECOMPRESSION ILLNESS. FINALLY A DIAGNOSTIC TEST TO CRY FOR?

Mike Bennett

Key Words
Bubbles, decompression illness, hyperbaric research.

Introduction

This presentation is intended to outline what may, in time, prove to be an important diagnostic tool for

decompression illness, examination of the tear film for small gas bubbles. It is not clear if this is going anywhere, but there are suggestions that such an examination at the time of presentation may prove clinically useful.

The diagnosis of decompression illness is not a straightforward proposition. In essence it is an historical diagnosis. The character, evolution and relationship of symptoms to the time and nature of the dive are of prime importance. Examination may contribute to diagnostic accuracy in some cases. We measure various neurological parameters, but quite often, there is very little to find on examination. There may be a role in some situations for measuring bubbles by a Doppler technique, but this is uncommon in the clinical setting.

There are other diagnostic tests that have been looked at over the years, and each has found its place in modern practice. For example, it may be useful to look at a chest X-ray if part of the differential diagnosis is pulmonary barotrauma. Clotting studies have been looked at from time to time around the Australasian hyperbaric centres. There are various scanning techniques, but these are more useful in the assessment of persistent symptoms and signs than as primary diagnostic tools. CT scanning, to take an example, is probably no more accurate as a diagnostic investigation than tarot cards, runes, and the favourite at our unit, swirling the tea leaves around while waiting for the patient to arrive.

Tear film bubbles

Why look for tear film bubbles? In 1670 Boyle observed bubbles in the aqueous humour of a viper's eye.[1] This was the first description of a bubble event in association with the eye. Little further interest was shown until 1978 when Simon and Bradley described the presence of bubbles under a hard contact lens after diving.[2] Socks, Molinari and Rowey first described bubbles under a gas-permeable contact lens in 1989 and later under a soft contact lens.[3] In 1991 Strath and Mekjavic described them under rigid centre, soft surrounding lenses.[4] It was pretty clear then, that if one wore contact lenses, there was some risk of developing bubbles underneath the lenses. This seemed to have no importance for people who were not wearing contact lenses.

Figure 1. Slit-lamp examination

In 1992 however, it was noticed that after repetitive dry diving, attending patients in a chamber, an attendant had bubbles in the eye, despite never having worn contact lenses.[5] That led to the work that we have done in an attempt to define any relationship between the presence of tear film bubbles and decompression stress.

Technique

The tear film is inspected through a standard slit-lamp (Figure 1) which is a standard tool for ophthalmologists and relatively easy to learn to use. However, unlike most slit-lamp examinations, we are not looking into the aqueous humour or at the cornea, iris and lens, but are primarily interested in the tear film.

The procedure with the slit-lamp is to sweep slowly from the medial to the lateral border of the inferior gutter (lower eyelid), counting bubbles, if any, as one goes. Such bubbles are often small and moving rapidly from lateral to medial within the gutter. It is important to limit one's inspection to the gutter itself in order to standardise the examination and because small bubbles on the lid itself are not uncommon, being the result of physical "foaming" after blinking. The subject is asked to close the eyes for 5 seconds, open them again and the examination repeated. Three sweeps are made and the bubble count averaged. This is a reasonably standard protocol as reported in the literature. Some centres have photographed the bubbles and made an estimate of bubble volume for each individual. It is not yet known if this significantly improves the accuracy of the procedure.

Tear film structure

The tear film is a surprisingly complex structure. The tear film's primary roles are to wet the globe, enable the lids to open and close with minimal friction and to reduce shearing stress on the globe itself. This contributes to the cornea remaining pristine and transparent. While there is clearly a film of tears over the whole globe, in the gutter along the bottom it is much thicker. The tear film flows surprisingly quickly from the outer to the inner canthus and down into the tear ducts.

When Strath et al. noted bubbles in the tear film of their chamber attendant, they did a reasonably simple study.[5]

They took 11 volunteers, compressed them in a chamber to 4 bar for 15 minutes and examined their tear films pre- and post-dive. Before compression, there was one individual with one bubble. The rest had none. After compression, there was an average of six bubbles in each eye examined (range 3-12) and this difference was statistically significant, $P<0.001$. They clearly demonstrated that before exposure there are very few bubbles indeed and that breathing compressed air increased the number of bubbles.

The origin of these bubbles became the focus of some further experiments, as did the possibility that bubble numbers may be related to decompression stress.

It remains unclear where the bubbles arise. There are a number of possibilities, based on the mechanisms by which the tear film is formed. The tear film is essentially a three layered structure (Figure 2). The innermost layer, close to the globe, is a mucin layer, which is composed of glycocalix and mucus produced by goblet cells on the cornea itself. Close to the globe this layer is quite thick and adherent to the cornea and becomes a more broken up and looser structure as one moves further out. This layer reduces much of the shearing stress. The next and most substantial layer is the aqueous, which is primarily produced in the lacrimal glands. The outermost layer is a thin layer of lipid which is produced by the destruction of Meibomian glands. These glands are very small and numerous, found on the lid margin, close to where we look at the film.

The Meibomian gland cells have a life span of three to four days, during which they swell with lipid. On maturity, they rupture into the ducts and the lipid is extruded to form a lipid layer, which stabilises the tear film. The film is therefore mainly aqueous, but with mucin at the bottom and the lipid layer stabilising it on the top.

Tear film bubble origin

Where the bubbles are formed is still not clear, but the likely possibilities have been discussed. They may evolve directly from the globe or the aqueous humour and this was the original concept used to account for these bubbles in 1992.[5] It is almost certainly the route by which bubbles under contact lenses are formed. Bubbles could also be derived directly by evolution from the conjunctival lid vasculature. It is argued that this possibility is supported by the observation that closing the eyes for some seconds seems to produce more bubbles than when they are kept open. Thirdly, bubbles could be formed in and secreted by the lacrimal gland. There does not seem to be any particular evidence for or against this possibility.

Alternatively, they could be introduced into the tear film with the rupture of Meibomian gland cells, and this we consider a strong possibility. The Meibomian glands are full of lipid and likely, when saturated, to contain considerable quantities of dissolved nitrogen. Further, this lipid is introduced to the tear film relatively late compared with the other elements of the tear film. The observed persistence of bubbles in the film for days following an episode of decompression stress may reflect the release of bubble-containing lipid into the film as Meibomian gland cells mature and rupture.

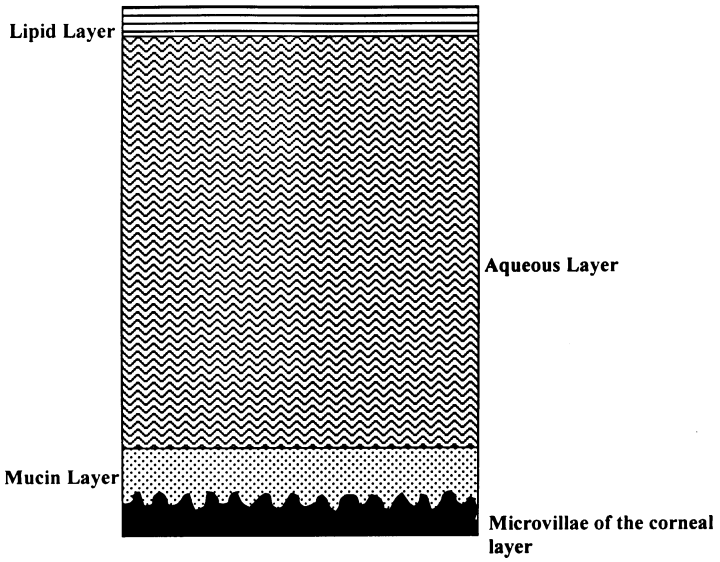

Figure 2. Composition of the tear film.

Bubbles and decompression stress

One of the early studies investigating bubble counts to was designed to relate the count to decompression stress. In a study published in 1998, evaluating the decompression stress of a standard diving table, Mekjavic et al. did bubble counts after dives to PADI no-stop limits with increasing bottom times.[6] They took 11 volunteers, subjected them to the compressions indicated in Figure 3 and measured the bubbles immediately on leaving the chamber. They also used a precordial doppler probe to detect any venous bubbles, however none were recorded during the study.

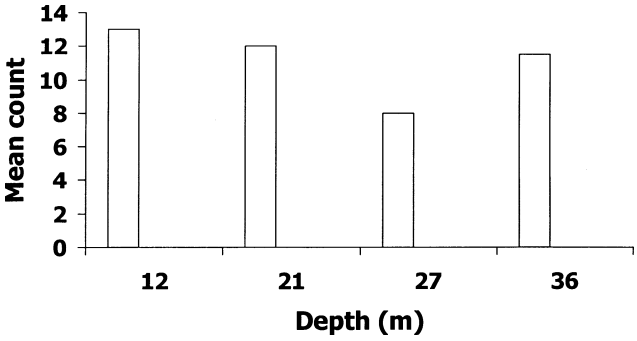

Figure 3. Bubble counts following no-stop limit compressions at increasing depth.[6]

The tear film bubble counts for each profile were essentially the same. The conclusion was that the decompression stress of these dives was roughly similar. As an addendum to that study, the 11 volunteers were compressed for increasing periods at 21 m (Figure 4). The bubble count rose with increasing decompression stress. Checking the statistics, which are not in the paper, there is a significant difference ($P < 0.05$) between the 15 and the 40 minute dives, but not between the 29 and the 40 minute dives.

About the same time, Morariu and his colleagues were looking at the effect of increasing nitrogen uptake on bubble generation by exercising during a dive.[7] They compressed 9 volunteers to 15 m for 15 minutes and 27 m for 15 minutes, exercising and non-exercising, and compared the bubble counts. The result for the 27 m compression was typical, 2.25 bubbles/subject at rest and 3.75 bubbles/subject after exercise. Mean bubble counts were consistently higher after exercising dives than resting dives, again implying that bubble counts are an index of decompression stress.

In all these studies bubble counts were taken immediately on leaving the chamber after the dive. While such findings are interesting, if bubble counts are to be of any use in the clinical setting, it is necessary to demonstrate that these bubbles persist for some time after diving. This is because frequently there is a significant time delay from diving to presentation. At the Prince of Wales Hospital, for example, the average time from the end of a dive to presentation is well over 24 hours.

To be clinically useful, in fact, the bubble count must persist for several days. In 1997, the first data was published in this area.[8] Six volunteers were compressed on two standard profiles, to 30 m for 15 minutes and 15 m for 180 minutes. The volunteers were examined every 24 hours for three days. Figure 5 shows the counts. It is this data which is used to support the supposition that Meibomian gland secretions are the source of the bubbles. There seemed no really good reason why these bubbles should persist, in essentially constant numbers, for two days after a dive, unless they were being stabilised in some way. Stabilisation within the fat of the Meibomian gland cell seems a likely candidate.

If bubble generation is related to decompression stress, it may follow that if one breathes oxygen rather than air, fewer bubbles will be detected. In 1997 Mekjavic tested this proposition.[9] Eight volunteers, did two simple 8 m, 60 minute chamber compressions, once on air and a week later on oxygen. Their tear films were examined before and after each dive. As one might predict after the air dive there was a significant increase in bubble count immediately after leaving the chamber, but when breathing oxygen there was a non-significant change in the number of bubbles. The pre-compression bubble counts in both groups were 3.0 per subject. Post-compression counts on air were 8.4 per subject and on oxygen 4.0 per subject.

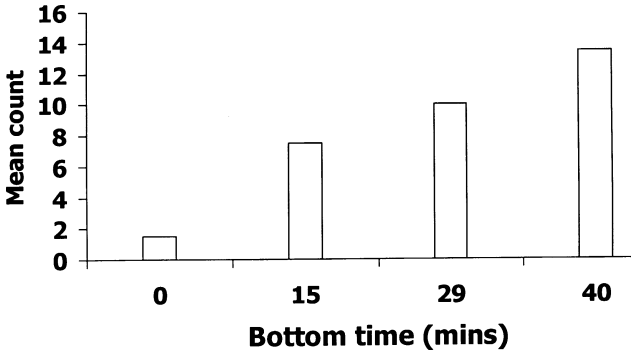

Figure 4. Bubble counts with increasing bottom time at 21m. $P<0.05$ for 15 minute v 40 minute compression.[6]

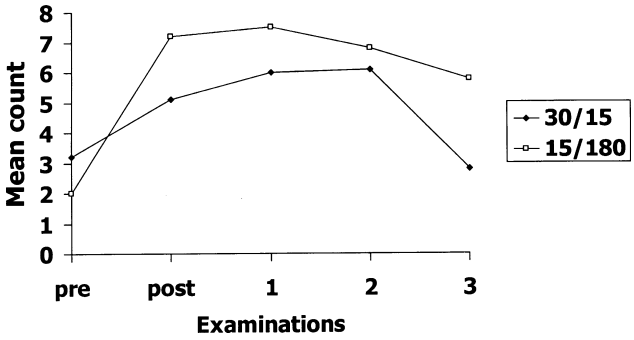

Figure 5. Bubble counts for 3 days following two dry chamber profiles.[8]

The future

To summarise our current state of knowledge in this area: bubbles can be detected in the tear film following dry chamber diving, they persist for a couple of days and seem related to decompression stress. Counts are increased by a number of things we would expect to increase decompression stress, bottom time, exercise and depth.

Is this observed phenomenon going to be of any use in clinical medicine? To begin to answer this question, a number of interim questions will need to be answered. Does one get the same phenomenon with real (wet) diving? Are counts sufficiently precise a phenomenon to distinguish between people who are really bent and those who are not? Is it going to be a useful measure to check the adequacy of treatment? After treatment and elimination of bubbles, should one re-examine the next day to check whether bubbles have returned, and if so, should one recompress?

At the Prince of Wales Hospital, we are attempting to answer some of these questions. At present, we are

interested in confirming that bubbles are detectable after wet dives and are halfway through a planned study in 60 volunteer divers. We are performing bubble counts for three days after a series of standard exposures in the ocean. In the future we intend to repeat that study with multi-day diving and see what sort of numbers we find. In addition, we use this examination for patients presenting with decompression illness to build a prospective data set for future analysis. We can report today that many of these patients have very high bubble counts on presentation, often as many as 20 or 30 on an average sweep.

All of this gives us some hope that finally we may be able to develop an accurate, highly predictive clinical diagnostic test for DCI that can be administered outside the hyperbaric facility and assist in making rational treatment and retrieval decisions. Time will tell, watch this space!

Audience Participation

Rhys Jones, New Zealand

Is it feasible to put slit-lamp microscope into the recompression chamber, and watch the bubbles form in dry dive volunteers?

Mike Bennett

Yes. We have not done it, although there is no reason why we should not. The equipment is on unofficial loan from the ophthalmology department, so we are being gentle with it. But there is no reason that I can see why we cannot do that. However slit-lamp examination is rather uncomfortable. It is a very bright light, but we have persuaded a couple of people to have a fairly lengthy examination. We have detected bubbles emerging from what we are pretty confident are Meibomian gland ducts. We are not experts in using slit-lamps, and these bubbles are notoriously evanescent. The bubble comes out, it appears, it washes down the tear film, and disappears. We attempt to get our ophthalmologists to come and look at them with us. Firstly they are incredulous that one would find bubbles. They say bubbles do not exist in their normal examinations, but of course, they do not usually look for them. When we do get them down and they see the bubbles, they instantly become interested in our discussions about bubbles, and they too feel, given the tear film structure and the nature of the bubble injury, that the Meibomian gland is a likely origin.

Rhys Jones

Have you tried different ascent rates?

Mike Bennett

No.

Richard Moon

When oxygen was given,[9] how did you give it? Was it with a head tent or mouthpiece or mask.

Bennett

That was not our study and the methodology was not reported.

Brubakk

I think they used a mask, but I am not sure.

I would like to make two comments. First, whether this can be used therapeutically, or to know if people have decompression sickness or not. I think that you will find, most likely, the same thing that we have found in vascular gas bubbles, namely that if there are many bubbles, the chance of having decompression illness clinical symptoms clearly increases. It seems that clinical risks are present above Grade 2, or 3 or 4 on the Spencer scale, and with more than 1.5 bubbles per cubic centimetre, if we actually measure bubbles in a vascular system. The more bubbles the more the risk of having clinical symptoms of decompression illness increases dramatically. To put it the other way, with a patient with vague symptoms, in whom you do not find any bubbles, then the likelihood of decompression illness is very low. It is our experience, and most other people's, that if one cannot find any bubbles at all, the diagnosis of decompression sickness is probably wrong. That is not always true, but one can find a lot of people, who have a lot of gas bubbles and no clinical symptoms. I think the major worth of this technique would be in the negative, to say if the symptomatology is dubious and if the exposure is dubious, and we cannot find any gas bubbles, then it is probably not decompression illness.

This has been shown in altitude decompression sickness with vascular bubbles. It is useful for taking out those who do not have decompression sickness.

My second comment relates to the lifetime of these bubbles. That is no surprise at all, because what happens can be shown theoretically and has been shown experimentally. With a significant number of bubbles, they mop up all the gas, and the gas tensions in the surrounding fluids become very, very low, which makes the gradient for removing the excess gas very low. So these bubbles have a very long lifetime. If they have a coating of lipid proteins that also increases diffusion resistance. Where they come from, we do not know, but all these bubbles grow from some sort of nuclei. So there must be a starting point for all these bubbles, they are not created, they grow from something. I think the Meibomian glands are a good candidate.

Mike Bennett

I agree with you on the likely usefulness. Even a highly negatively predictive test would be of use. As you know, we quite often have to transport our divers a long way, on a pretty dubious set of symptoms and a normal examination. A slit-lamp is equipment which is pretty freely available in Australia. One could get a potential candidate for treatment, who is now 36 hours after some pretty

innocent diving with some vague symptoms, competently examined with a slit-lamp and find there are no bubbles. Perhaps we would then be in a position to say "Let us save the country $30,000, and not fly him down to Sydney."

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GLEANINGS FROM MEDICAL JOURNALS

HYPERBARIC OXYGEN

A role for oxygen-induced osmosis in hyperbaric oxygen therapy.
Hills BA.
Med Hypotheses 1999; 52: 259-263

Abstract

The principles of gas-induced osmosis, demonstrated in the 1970s, have been applied to the very large steady-state gradients of O_2 arising between arterial blood and hypoxic tissue during hyperbaric oxygen (HBO) therapy to produce a fluid "pump" in the desired direction for resolving accompanying oedema. Thus, in soft-tissue injuries, an oxygen-induced fluid pump would break the vicious cycle between ischaemia, hypoxia and oedema at the point of oedema rather than hypoxia, as hitherto assumed. This osmotic mechanism enables the successes of HBO therapy in hypoxic disorders to be reconciled with early failures in such areas as hyperbaric radiotherapy, where substitution of O_2 for N_2 in inspired air was clearly not reflected at the tissue level. This argument also applies to the success of HBO in treating air embolism and decompression sickness over simple compression. The oxygen pump would seem to offer a more plausible explanation for the success of HBO therapy than theories based upon O_2 delivery by the circulation, especially when considering cardiovascular reflexes to elevated PaO_2 and the marginal increase in blood O_2 content upon switching to HBO from normobaric oxygen breathing.

Key Words
Hyperbaric oxygen, physiology, reprinted, treatment.

PULMONARY BAROTRAUMA

Diving and the risk of barotrauma
Russi EW
Thorax 1998; 53 (suppl) 2: S20-S24

Abstract

Pulmonary barotrauma (PBT) of ascent is a feared complication in compressed air diving. Although certain respiratory conditions are thought to increase the risk of suffering PBT and thus should preclude diving, in most cases of PBT, risk factors are described as not being present. The purpose of our study was to evaluate factors that possibly cause PBT. We analysed 15 consecutive cases of PBT with respect to dive factors, clinical and radiologic features, and lung function. They were compared with 15 cases of decompression sickness without PBT, which appeared the same period. Clinical features of PBT were arterial gas embolism (n = 13), mediastinal emphysema (n = 1) and pneumothorax (n = 1). CT of the chest (performed in 12 cases) revealed subpleural emphysematous blebs in 5 cases that were not detected in pre-injury and post-injury chest radiographs. A comparison of pre-dive lung function between groups showed significantly lower mid-expiratory flow rates at 50 and 25% of vital capacity in PBT patients ($P < 0.05$ and $P < 0.02$, respectively). These results indicate that divers with pre-existing small lung cysts and/or end-expiratory flow limitation may be at risk of PBT.

Key Words
Decompression illness, pulmonary barotrauma, reprinted, risk.

DIVING ACCIDENTS

Open water scuba diving accidents at Leicester: five years' experience.
Hart AJ, White SA, Conboy PJ, Bodiwala G and Quinton D.
J Accid Emerg Med 1999; 16: 198-200

Abstract

The aim of this study was to determine the incidence, type, outcome and possible risk factors of diving accidents in each year of a 5-year period presenting from one dive centre to a large teaching hospital accident and emergency (A&E) department. All patients included in this study presented to the A&E department at a local teaching hospital in close proximity to the largest inland diving centre in the UK. Our main outcome measures were: presenting symptoms, administration of recompression treatment, mortality, and postmortem examination report where applicable. Overall, 25 patients experienced a serious open water diving accident at the centre between 1992 and 1996 inclusive. The percentage of survivors (n=18) with symptoms of decompression sickness receiving recompression treatment was 52%. All surviving patients received medical treatment for at least 24 hours before discharge. The median depth of diving accidents was 24 m (range 7-36 m). During the study period, 1992-96, the number of accidents increased from 1 to 10 and the incidence of diving accidents increased from four per 100,000 to 15.4 per 100,000. Over the same time period the number of deaths increased threefold. The aetiology of the increase in the incidence of accidents is multifactorial.

Important risk factors were thought to be: rapid ascent (in 48% of patients), cold water, poor visibility, the number of dives per diver and the experience of the diver. It is concluded that there needs to be an increased awareness of the management of diving injuries in an A&E department in close proximity to an inland diving centre.

Key Words

Accidents, deaths, decompression illness, recreational diving, reprinted, risk, treatment.

CARBON MONOXIDE

British Hyperbaric Association carbon monoxide database, 1993-96.

Hamilton-Farrell MR.

J Accid Emerg Med 1999; 16: 98-103

Abstract

To study the referral pattern of patients, poisoned with carbon monoxide and subsequently transferred to British hyperbaric oxygen facilities, from April 1993 until March 1996 inclusive. A standard dataset was used by hyperbaric facilities within the British Hyperbaric Association. The data on each patient were sent in confidence to the Hyperbaric Unit at Whipps Cross Hospital for analysis. The epidemiology of poisoning and the population studied were analysed. Times of removal from exposure, referral to a hyperbaric facility, arrival at the hyperbaric facility and start of treatment were recorded. Data on the outcome of the episode were documented in one of the contributing facilities. 575 patients exposed to carbon monoxide were reported as being referred to British hyperbaric facilities in the three years, the busiest facilities being in London and Peterborough. The proportions of accidental and non-accidental exposures were 1: 1.05. Of the accidental exposures, central heating faults were responsible in 71.5% of cases (n = 206). Smoke inhalation from fires was responsible for a further 13.5% (n = 39). The mean delay to arrival in a hyperbaric oxygen facility was 9 hours and 15 minutes after removal from exposure. Recovery after treatment was sometimes incomplete. The reported pattern of referral was regionally weighted towards the south east of England. Smoke inhalation victims were often not referred for hyperbaric oxygen treatment. The delay to treatment was multifactorial; and the mean delay was well in excess of six hours. There is room for improvement in the consistency and speed of referral. Treatment schedules require standardisation. A central advice and referral service would be helpful.

Key Words

Carbon monoxide, hyperbaric oxygen, data, reprinted, risk, sequelae.

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Unit 2	10/7/00	to	14/7/00
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Unit 3	23/10/00	to	27/10/00

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