The Editor's Offering

Readers will have noticed that the cover of the Journal, which has been unchanged since it was introduced in March 1989, has changed its format. There are two reasons for this break with what can probably be called a tradition as it has endured for 11 years. The more important one is that people, who are not Australasian divers interested in diving safety or diving medicine, are unaware of what SPUMS stands for. The other is that one of the Indexing organisations has, among many things, in its list of factors which influence it towards accepting a journal that the title of the publication clearly reflect its subject matter. The two together decided the Committee to change the name from SPUMS Journal to South Pacific Underwater Medicine Society Journal. However the Committee bear no responsibility for the final layout of the new titling arrangements which have been tinkered with up to the time this is being typed into the Editor's Macintosh computer. Reader feed back will be appreciated even if not acted upon!

At the 2000 Annual General Meeting in Fiji, which finished a week before the coup, a motion was passed to change the name and composition of the Board of Censors, those volunteers who supervise the standards of medical education and experience which are required for those members who aspire to the SPUMS Diploma in Diving and Hyperbaric Medicine (DDHM) or to run courses in Diving and Hyperbaric Medicine to cover the education required by doctors before they undertake diving medicals. The various alterations which have to be made to the Purposes and Rules are printed on page 86. If any member disagrees with these changes he or she should write to the Secretary of SPUMS objecting before September 1st 2000. If no objections are received it will be assumed that the membership approves of the changes. The new body will be the Academic Board and its constitution is laid out on page 86.

Dr Douglas Walker's 1997 report on diving-associated deaths in Australia highlights a remarkable rise in the number of people who died while snorkelling to 17 in the year. Only four scuba divers and two hose supplied divers died in 1997, approximately one third as many as those who died snorkelling. Presumably these deaths are in part due to the advanced years and poor health of many tourists to the Great Barrier Reef and their lack of experience with a snorkel. The Editor remembers quite clearly the first time he used a snorkel, in 1973, and his difficulty in adjusting to breathing through his mouth.

Dr Tony Holley provides a validation of the Royal New Zealand Navy (RNZN) decompression illness (DCI) scoring system (published in the Journal in 1998; 28 (2):84-94). This retrospective study shows that those whose admission score is 25 or less have an approximately 90% chance of being cured, that is discharged without sequelae, with treatment. The RNZN system allows for accurate daily assessment. The Journal congratulates Dr Holley on the award of his DDHM.

Dr Geoff Taylor, whose paper on the heat treatment of fish spine injuries appeared in the last issue, moves on to jellyfish stings. He has discovered, by personal experience, that heat treatment removes the pain from, at least some, jellyfish stings. Here is a well watered field for some research in Eastern Australia, where the jellyfish may perhaps be different from Western Australian waters.

Dr Carl Edmonds is back with Diving Doctor's Diary, featuring carpal tunnel syndrome masquerading as DCI. One of his references is off the internet and we have provided the, very long, address so that readers may find the paper which is, as of yesterday, not available, using the normal search methods, to the Librarian at the Australian and New Zealand College of Anaesthetists (ANZCA).

This issue has good news for those who wish to take part in the ANZCA Special Interest Group (SIG) in Diving and Hyperbaric Medicine on page 88. The reason that they have not heard from the SIG is that the ANZCA secretariat has mislaid their names and addresses. Please register your interest in the SIG again as arrangements have been made not to lose names and addresses!

The papers from the 1999 Annual Scientific Meeting cover the fundamental question of what is the right treatment dose for DCI (Prof Alf Brubakk) and the position and prospects for using drugs to assist in the treatment of DCI with pressure and oxygen (Prof Richard Moon). Dr Chris Acott provides a gloomy picture of the sequelae suffered by South Australian tuna farm divers after treatment. Dr Robyn Walker provides two case reports which demonstrate that exercise is not always good for you, especially if you have trained yourself to tolerate high carbon dioxide levels to increase your underwater endurance. Cautionary tales which, perhaps, justify the Editor's dislike of exertion.

The most interesting of the reprinted articles is that by Hamilton and Baker on the recent Reverse Dive Profiles Workshop. A review of the printed workshop proceedings will appear in the next issue. From Britain we reprint a review of the report *Scuba Diving: A Quantitative Risk Assessment* produced by the Health & Safety Executive in 1997 and a summary of the 1999 *Diving Incidents Report* of the British Sub-Aqua Club.

ORIGINAL PAPERS

AUSTRALIAN DIVING-RELATED DEATHS IN 1997

Douglas Walker

Key Words
Accidents, deaths.

Summary

This report records the details of deaths referred to Coroners as having occurred while the deceased had been either swimming or diving using a snorkel or was diving using compressed air. It is highly probable that not all fatalities occurring during such activities have been identified but the information provides a factual basis for discussion of how to reduce such fatalities. The data for 1997 is unusual in the number of snorkel users who died and the fact that all save one occurred in visitors to the Great Barrier Reef region. The presumed reason is that only there is a commercial interest in providing facilities for snorkelling and a corresponding Governmental interest in any fatalities which occur. There were seventeen (17) snorkel deaths identified and eleven (11) of these were overseas visitors. Only one death (BH 97/6) was outside the Great Barrier Reef area. Ill health (cardiac) was the critical factor in many instances. Only four (4) deaths were identified among scuba divers and two (2) using surface supply.

Snorkel swimmers and breath-hold divers

Of the seventeen fatalities identified, a cardiac factor was either a probable or certain critical factor in nine.

Case BH 97/1

This man had a history of an episode of tachycardia some five years before and was taking medication to prevent a recurrence. However he had reported no ill health since this episode. As his wife destroyed all his tablets after his death it is not known what he was taking. He went snorkelling alone after booking to join a conducted snorkel swim in the afternoon. The morning swim which he had intended to join had been cancelled because of expected rough water. Later his wife and daughter walked along the beach and found his clothing, so they assumed he was still swimming and would return later. When he failed to join them at the midday meal they became worried, the more so when they heard an announcement calling for a doctor. Shortly after this they were informed that one of the staff had noticed something in the water about 100 m off the beach. He thought it was a turtle caught in a mooring rope but when he swam out he discovered it was a body. Although his left arm was apparently tangled in the rope this had not apparently been the reason he drowned. It is assumed that he probably suffered an episode of cardiac tachycardia and the resulting incapacity led to his drowning, though this cannot be proved.

SOLO SWIM USING SNORKEL. HISTORY OF TACHYCARDIA FOR WHICH HE TOOK MEDICATION. PROBABLE CAUSE DEATH WAS CARDIAC ARRHYTHMIA LEADING TO DROWNING.

BH 98/2

The sudden death of this apparently healthy young woman without warning was particularly unexpected because she had recently passed a “diving medical” check and just completed a scuba diving course. A bonus scuba dive was offered to them on completing the course but she decided to snorkel swim instead. This decision may have been because she wished to take some underwater photographs without any interruptions. She was with the group of scuba divers but no special attention was paid to her while two of the group were being towed closer to the reef where they were to dive, following which another diver requested attention. She was seen floating upright, head and shoulders out of the water, near the channel marker. She appeared to be signalling to be picked up. It was then slack tide and there was no significant current running. When the dive boat reached her she was floating as if snorkelling, then rolled over and her head became submerged but she failed to react. She was pulled aboard and CPR commenced but failed to show any response. Histology evidence of idiopathic lymphocytic myocarditis was found following the autopsy.

SOLO SNORKEL. CALM WATER. SILENT SURFACE DEATH. RECENT DIVING MEDICAL. JUST COMPLETED SCUBA COURSE. MYOCARDITIS.

BH 97/3

While on holiday in Australia this man visited one of the island resorts. There he met other visitors, some of whom asked him to join them snorkelling, but he declined as he had no equipment. However he decided to follow their example the next day but did not join them as they were intending to scuba dive. He hired a mask and snorkel, but no fins, and went to a beach and there teamed with two others. He was not as strong swimmer as they were. After 20 to 30 minutes travelling with the current he decided to return to their starting point but the other two chose to continue longer over a reef. When they last saw him he appeared to be snorkelling in a confident manner, but shortly

afterwards they heard a cry for help and saw him waving his arms above his head. He was unconscious and sinking by the time they reached him. They brought him ashore as quickly as possible, a difficult task as here the shore was sharp rocks. The drowning was thought to be due to fatigue, with his poor physical condition, poor swimming ability and lack of fins all contributing.

NOT USING FINS. SEPARATED. THEN SURFACE SNORKEL SWIM AGAINST CURRENT. POOR SWIMMER. EXPERIENCE UNKNOWN.

BH 97/4

Although he had had a triple coronary bypass in 1990 he did not seek medical during his visit to Australia when he experienced an episode of faintness, loss of vision and pain in the back of his head while hiking in the Blue Mountains three weeks before he and his wife visited the Barrier Reef on a live-aboard boat. Although there were other passengers swimming and snorkelling in the water, he chose to snorkel with his wife away from the others. The safety boat came and offered to take them back to the main group but this offer was declined. However, the boat returned a short time later and he then suggested that his wife be towed, then he was seen floating face down and apparently lifeless a short distance from her. Resuscitation efforts were unsuccessful.

SURFACE SNORKEL CLOSE TO WIFE. CALM WATER. SILENT RAPID DEATH. SAFETY BOAT CLOSE BY. HISTORY OF CORONARY ARTERY BYPASS 7 YEARS BEFORE. EPISODE OF ILL HEALTH WHILE WALKING 3 WEEKS BEFORE DEATH. ACUTE CARDIAC DEATH.

BH 97/5

Two couples from overseas were on holiday on a reef island and decided to snorkel off a beaches. It was calm and shallow and there were already about twelve others on the beach and five in the water when they arrived. As they had brought only two sets of equipment with them it was decided the two men would use it first, then the two women. However one of them had persistent trouble with water entering her mask so returned to shore, telling her friend she would get one of the men to take her place. As they could see her swimming strongly from where they were sitting on the beach there was no hurry in the hand over. It was about five minutes before they looked again and then they were unable to see her so decided to walk along the beach, expecting to find that she had come ashore further up the beach. They came across a group giving CPR to a person who had been found floating face down in the shallow water near the beach. Although she responded to resuscitation she died in hospital the next day from the effects of the cerebral anoxic damage. No health factors were found to explain why she drowned. Possibly she inhaled some water and panicked, failing to stand up in the waist deep water. There is no information as to whether she had any snorkelling experience but she had appeared to have no trouble snorkelling when last seen.

SEPARATION. THEN SOLO SNORKEL. CALM SHALLOW WATER. CLOSE TO OTHERS. SILENT INCIDENT. DELAYED DROWNING DEATH.

BH 97/6

Two divers were spear fishing near the harbour entrance. After about an hour and fifteen minutes the buddy left the water but the other diver continued. About 15 minutes later the buddy saw two fishing boats enter the shipping channel and then saw his friend surface in their path and fail to reappear after they had passed. He realised his friend must have been hit so rushed to the harbour to ask the fishermen to return, then entered the choppy water to search for him. The body was not recovered for a further 1.5 hours, with two head wounds.

SPEARFISHING. SEPARATED SO SOLO. SURFACED IN BOAT CHANNEL IMMEDIATELY IN FRONT OF TWO FISHING BOATS. NO DIVERS FLAG. FATAL HEAD INJURIES FROM PROPELLER.

BH 97/7

An optional extra for passengers on this cruise ship was a day trip to visit the Barrier Reef and this couple were among the large group which signed up. They were taken out to a pontoon, permanently moored at one of the reefs, and each given mask, fins, a snorkel and a talk on the use of this equipment. The victim admitted to being a poor swimmer and was provided with a life jacket before he entered the water. There were two crew deputed to watch over those in the water as they swam around using their snorkels. One noticed that the victim was floating motionless, face down, and raised the alarm before swimming out to check what was wrong. He was soon joined by the safety dinghy. Their resuscitation efforts were unavailing. Although there was no history of ill health the autopsy showed the presence of severe coronary atheroma and evidence of an area of myocardial scarring. This probably indicates that his history of recent “indigestion” may have been unrecognised angina. The circumstances indicate that this should be regarded as death from cardiac causes.

SNORKELLING IN GROUP. SILENT RAPID DEATH. EFFICIENT SUPERVISION. NO HISTORY OF ILL HEALTH. CORONARY ARTERY ATHEROMA. CARDIAC DEATH.

BH 97/8

Among the passengers on this trip out to the Barrier

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and Experience		Dive	Dive	Depth in metres		Weights	
		Victim	Buddy	Group	purpose	Water	Incident	On	kg
BH 97/1	73	Training and experience not stated	No buddy	Solo	Recreation	Not stated	Surface	None	None
BH 97/2	29	Training and experience not stated	No buddy	Solo	Recreation	Not stated	Surface	None	None
BH 97/3	28	Training and experience not stated	Training and experience not stated	Group Separation before incident	Recreation	3 m	Surface	None	None
BH 97/4	67	Some training No experience	Training and experience not stated	Buddy Separation before incident	Recreation	Not stated	Surface	None	None
BH 97/5	56	No training No experience	No training No experience	Buddy Separation before incident	Recreation	1 m	Surface	None	None
BH 97/6	32	No training Experienced	No training Experienced	Buddy Separation before incident	Spear fishing	9 m	Surface	On	Not stated
BH 97/7	75	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 97/8	31	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 97/9	43	No training No experience	Trained Experienced	Group No separation	Recreation	Not stated	Surface	None	None
BH 97/10	72	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 98/11	60	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 97/12	46	No training No experience	No training No experience	Group Separation before incident	Recreation	Not stated	Surface	None	None
BH 97/13	46	No training No experience	No training No experience	Buddy Separation before incident	Recreation	1 m	Surface	None	None
BH 97/14	67	Training and experience not stated	Trained Experienced	Group No separation	Recreation	Not stated	Surface	None	None

DIVING-RELATED DEATHS IN 1997

Buoyancy vest	Contents gauge	Remaining air	Equipment Tested	Equipment Owner	Comments
None	Not applicable	Not applicable	Not applicable	Hired	Episode of tachycardia 5 years before. Rough sea so not group dive Cardiac death.
None	Not applicable	Not applicable	Not applicable	Hired	Sudden death. Apparently healthy Myocarditis
None	Not applicable	Not applicable	Not applicable	Own	First use of snorkel. No fins. Not strong swimmer. Swam into current, fatigued.
None	Not applicable	Not applicable	Not applicable	Hired	Coronary bypass 7 years before. Acute cardiac death.
None	Not applicable	Not applicable	Not applicable	Not stated	Separation then solo. Calm water. Unknown swimming, and snorkel ability.
None	Not applicable	Not applicable	Not applicable	Own	Surfaced in boat channel. Hit by propeller.
Life jacket	Not applicable	Not applicable	Not applicable	Hired	First use of snorkel? Poor swimmer. Wore life jacket. Silent death in crowd.
None	Not applicable	Not applicable	Not applicable	Hired	First use of snorkel. Calm sea. Silent surface problem. Delayed drowning death.
None	Not applicable	Not applicable	Not applicable	Hired	Obese diabetic became breathless. Towed but died. Acute cardiac failure.
None	Not applicable	Not applicable	Not applicable	Not stated	Parkinsonism. Good swimmer. Asymptomatic coronary disease.
None	Not applicable	Not applicable	Not applicable	Hired	History of previous myocardial infarction. Inadequate surface watch. Current. Cardiac death.
None	Not applicable	Not applicable	Not applicable	Hired	No fins? Poor fit mask, snorkel? Silent surface death. Excess alcohol a factor.
None	Not applicable	Not applicable	Not applicable	Hired	First use of snorkel? Collapsed standing on reef. Renal disease, cardiac factors.
None	Not applicable	Not applicable	Not applicable	Hired	History of coronary artery bypass.

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training and Experience		Dive	Dive	Depth in metres		Weights	
		Victim	Buddy	Group	purpose	Water	Incident	On	kg
BH 97/15	72	Trained Experienced	Trained Experienced	Group Separation before incident	Recreation	18 m	Surface	On	3 kg
BH 97/16	33	No training No experience	No buddy	Solo	Recreation	3 m	Not stated	Not stated	Not stated
BH 97/17	24	Training and experience not stated	Training and experience not stated	Buddy Separation before incident	Spear fishing	27 m	Not stated	Off	4.5 kg
SC 97/1	38	Trained Experienced	Trained Experienced	Buddy Separation during incident	Recreation	40 m	40 m	On	12 kg
SC 97/2	58	Trained Experienced	Trained Experienced	Buddy Separation before incident	Recreation	32 m	Surface	On	Not stated
SC 97/3	43	Trained Some experience	Trained Experienced	Group Separation before incident	Recreation	18 m	Surface	On	9 kg
SC 97/4	47	Trained Some experience	Trained Experienced	Group Separation before incident	Deep dive course	30 m	Ascent	Buddy ditched weights	14 kg
H 97/1	40	Some training Experienced	No training Experienced	Buddy Separation before incident	Cray fish	3 m	Ascent	On	Not stated
H 97/2	30	Trained Experienced	Trained Experienced	Buddy Separation before incident	Spear fishing	36 m	Ascent	On	Not stated

Reef was a group of overseas visitors with their tour guide. He translated to them the talk on snorkel diving which was routinely given during the outward trip. While members of the group, and other passengers, were swimming near the pontoon, one of them noticed the victim floating immobile, face down, about 10 m from the pontoon. The snorkel was out of her mouth and there was some water in her mask. The safety watcher quickly swam out and retrieved the victim and commenced CPR, which was rewarded with a resumption of breathing after 30 minutes. She died in hospital 4 days later from the effects of anoxic brain damage. Nothing is known concerning her swimming ability, personality, or possible previous use of a snorkel. The water was calm and there were 10-20 other swimmers nearby but none were aware she was in trouble.

SNORKEL SWIMMING IN CROWD. CALM WATER. SILENT INHALATION OF WATER. PROLONGED CPR BEFORE RESPONSE. DELAYED DROWNING DEATH

BH 97/9

Two friends, on holiday from overseas, joined a day trip to the Barrier Reef. Although one of them was obese and a diabetic she admitted to no ill health when signing on to go snorkelling. The passengers were shown a video on snorkelling and were given a talk. The two friends had lunch after spending some time snorkelling near the pontoon and then joined a guided tour of an adjacent reef after demonstrating their snorkelling ability to the guide. This was a requirement because the tour passed over deep water and lasted 45 minutes. The victim was noticed to swim slower than the others but declared she was not feeling

DIVING-RELATED DEATHS IN 1997

Buoyancy vest	Contents gauge	Remaining air	Equipment		Comments
			Tested	Owner	
None	Not applicable	Not applicable	Not applicable	Own	Inadequate safety watch. Rough water. Polycystic kidneys. Coronary atheroma.
None	Not applicable	Not applicable	Not applicable	Borrowed	Solo. No medical history available. Body never recovered.
None	Not applicable	Not applicable	Not applicable	Own	Post-hyperventilation type death.
Not inflated	Yes	None	OK	Own	'Silt out' in wreck.
Not stated	Yes	+++	OK	Own	Surface swim to start dive. Cardiac death?
Not inflated	Yes	+++	OK	Own	Separation on descent. Unexplained drowning.
Not inflated	Yes	+++	OK	Hired	Separation during class ascent. Nitrogen narcosis effect? CAGE.
None	No	None	Faults	Own	Supply hose separated. Out-of-air ascent. Panic ascent. CAGE.
None	No	None	Serious faults	Borrowed	Compressor failed. Lost air. Out-of-air ascent. CAGE.

unwell. However, after 25 minutes she was noted to be very breathless and she accepted an offer to tow her back to the boat. Before this could be done she became unconscious and ceased breathing. She was brought back to the boat quickly. CPR was appeared to be unsuccessful but she was transported by helicopter to the base hospital where some return of vital signs was noted, but not maintained. This was diagnosed as death from acute cardiac failure due to arrhythmia. The pathologist noted the presence of moderate coronary and patchy myocardial fibrosis. It was suggested that she was probably unused to making the strenuous exertion required during this swim against a current. As is often the case in drowning, resuscitation efforts were made more difficult by regurgitation of fluids.

SNORKEL SWIM IN GROUP. BECAME ACUTELY BREATHLESS THEN UNCONSCIOUS. OBESE DIABETIC. MODERATE CORONARY ATHEROMA AND PATCHES OF MYOCARDIAL ISCHAEMIC DAMAGE. PROBABLY CARDIAC ARRHYTHMIA CAUSED ACUTE CARDIAC FAILURE.

BH 97/10

This man was visiting his son, who had a boat. They decided to take it out to a local reef and go snorkelling. The father made a backward roll entry into the water and then swam back to hold onto the boat. He mentioned that he had swallowed a lot of water but was cheerful and appeared well. When his son entered the water half a minute later he was surprised to see him floating face down near the boat. His CPR was unsuccessful. There was no history of ill health beyond some Parkinsonism symptoms. The autopsy showed marked coronary atheroma with severe stenosis of the right

anterior coronary artery. The cause of death was given as being cardiac arrhythmia combined with inhalation of sea water. There was no history of cardiac symptoms and he was not taking any medications. He was said to be a good swimmer.

SUDDEN SILENT DEATH AFTER WATER ENTRY FROM BOAT. SOME ASPIRATION OF WATER REPORTED BUT APPEARED UNAFFECTED. SOME PARKINSONISM SYMPTOMS. CORONARY ATHEROMA PRESENT. CAUSE OF DEATH CARDIAC ARRHYTHMIA AND INHALATION OF SEA WATER.

BH 97/11

During the trip out to the Reef this couple, both of whom had made 3 or 4 previous snorkel dives, were exposed to a safety talk concerning boat procedures, snorkelling, and swimming abilities. The wife later claimed not to have heard any such talk. Getting the active attention of passengers cannot be guaranteed. Following a trip in the glass bottomed boat the couple went snorkelling away from others who were swimming or snorkelling near the boat while other passengers were scuba diving or on the cay's beach. The couple found they could not swim back to the cay against the current and his wife was pleased to be assisted back to shore by two of the other passengers who responded to her husband's signals. They later said they did not see him when they arrived to assist her. Once ashore she became aware of her husband's absence and he was then seen in the water and appeared to be experiencing some difficulties. When reached by two others of the passengers he was floating face down, limp and probably dead. There was some delay before the skipper was informed that some problem had occurred and brought over the glass bottomed boat. CPR was begun as soon as he was pulled aboard but there was no response. Autopsy revealed that he had suffered a myocardial infarct and there was evidence of a previous one. There was marked coronary atherosclerosis. His wife was unaware of him having any health problems apart from "some blood pressure, for which he occasionally took half a tablet". It is very tempting to assume that the tablet was for angina, but there is no corroboration of this supposition. Although there was no efficient safety watch from the beach, this did not effect the outcome in this instance.

SILENT SURFACE DEATH SHORTLY AFTER SEPARATION FROM OTHERS. STRONG SURFACE CURRENT. AUTOPSY SHOWED PREVIOUS UNKNOWN MYOCARDIAL INFARCT. FATAL FURTHER INFARCT.

BH 97/12

There is uncertainty whether this man was free from the influence of the previous night's drinking when he boarded the boat on a day to The Reef. Similarly there were differing opinions as to whether passengers were advised to swim in buddy pairs. Although he was issued with mask, snorkel and fins there is no certainty that he was actually wearing these when he was last seen close to the boat after entering the water. His intended buddy decided to abort his swim soon after entering the water and the victim then swam away without his actions being noticed. He was later found floating face down by the crew of another boat who noticed his failure to react to the wash of another boat passing over him. His mask, snorkel and fins were apparently missing when he was reached. No medical reason was found for his drowning in these circumstances, but perhaps this was his first use of a snorkel and he panicked when water entered his snorkel.

SILENT SURFACE DEATH AFTER SEPARATION FROM GROUP. SNORKEL USE EXPERIENCE UNKNOWN. EQUIPMENT MISSING SO UNCERTAIN WHETHER WORN.

BH 97/13

Two women friends, on holiday from overseas, joined a day trip to visit one of the reefs. During the trip out the safety talks were given in several languages though later questioning showed that there was poor recall of mention of health factors. After arriving at a sandy cay they were ferried ashore Lifejackets were made available to those who desired them, and masks, snorkels and fins were given to those who wished to do more than simply swim. The two women began snorkelling close together but soon increased their separation as they kept bumping into each other. The friend had snorkelled previously and helped the victim get ready, then watched her closely until satisfied that she was managing without any problems. There was some chop but the water was clear, there were others nearby and a safety watch was maintained from the beach. The friend noticed the victim standing unsteadily on the reef in waist deep water, her mask off, waving her arms. The buddy joined her and tried to steady her but failed and she toppled forwards into the water. Several nearby swimmers and a crew member quickly came to help. EAR was commenced while in the water and continued after she was brought back onto the boat but she did not respond. It was later found that she had a history of renal disease and had a poor effort tolerance, becoming breathless while walking except on level ground. Cause of death was given as drowning but the presence of amyloid disease and myocardial fibrosis changes indicates that this was a cardiac death.

FIRST USE SNORKEL. SHORT SEPARATION. THEN STOOD ON REEF IN SHALLOW WATER WAVING FOR ASSISTANCE. COLLAPSED AND DIED. SEVERE ILL HEALTH. RENAL AND CARDIAC DISEASE. FAILED TO RECOGNISE THE EFFORT DEMANDS OF SWIMMING WITH SNORKEL.

BH 97/14

This day trip to the reef was run in the regular manner, with talks on the boat's emergency procedures and giving basic instruction on snorkelling to those intending this activity. The victim, a visitor from overseas, declared his history of a myocardial infarct in 1984 and that he had suffered no subsequent ill health. His only medication was a daily dose of aspirin. The reasonable decision was taken that he could join the guided snorkel tour of the reef. There were two groups, one of the younger passengers, the second of the more elderly. He was included in the latter group. The group leader made frequent stops and swam slowly in appreciation of this group's probable abilities. After about 15 minutes the victim was noticed to be tired, though he made no complaint, so the leader decided to tow him back to the dinghy which would return him to the boat. He was too breathless by the time they reached the dinghy to climb into it without assistance, so the OxyViva was used, then he lost consciousness and CPR was initiated. He was airlifted to hospital but died later that day. Autopsy revealed a fresh myocardial infarct and evidence of his previous one. At no time did he mention pain, only tiredness and breathlessness.

RAPID DEVELOPMENT OF BREATHLESSNESS DURING SUPERVISED SLOW SNORKEL SWIM. HISTORY OF PAST MYOCARDIAL INFARCT (1984). FATAL FURTHER MYOCARDIAL INFARCT. DIVE LEADER GAVE CORRECT AND RAPID MANAGEMENT OF THE SITUATION.

BH 97/15

The investigation of this fatality was deliberately, but perfectly legally, degraded by the natural desire of those most involved to avoid possible self-incrimination, and although their response may have been excessive it was effective in reducing the availability of important data and has make it impossible to clarify some inconsistencies in the evidence which is available. It is agreed that adverse sea conditions had caused the cancellation of several previously arranged snorkel and scuba dives in the days preceding this dive, and that the conditions were such that only those who claimed to be experienced snorkel divers were to be taken out on the boat. The dive boat carried a skipper and a diving instructor, who had two pupils among the 15 or so snorkel divers aboard. Two smaller boats followed, one with an additional 5 snorkel divers, the other with several scuba divers. There was no apparent check on the real experience of the snorkel divers, several of whom appear to have considered that their scuba diving experience placed them in this category. They were instructed after entering the water to swim to the reef edge and then the current would take them to a bommie and where the dive boat would pick them up 40 minutes later. No instructions were reportedly given concerning the dive location (one witness contradicted this) or advice to follow buddy system practices and no adequate surface safety watch was arranged. The sea conditions differed from those expected, a strong current being present, a fact not noticed by the skipper until it was observed that the members of the group were scattered and being carried away from their intended destination and were signalling to be picked up. One of them later claimed to have drawn attention to a diver in need of assistance, the victim, but the boatman insisted in picking up another pair before going to him, and other witnesses confirmed this.

They had entered the water about 50 m from the reef and to reach the bommie they were intending to drift some 200-300 m. The water was 20 m deep where they entered the water. They had been advised to attract assistance by raising one or both arms and waving them, an action which often results in the person becoming submerged. There is some uncertainty concerning the sea conditions, variously described as half a metre to 6 foot swell. But the current was certainly contrary in direction to that they had been expecting, and rated as being strong by most of them.

When the boat reached the victim he was face down, "vertical in the water with the top of his head 10-20 cm below the surface". He was quickly pulled into the boat and EAR commenced, although he appeared to be dead. One witness described the divemaster as panicking and performing inefficient EAR, but this is unlikely to have affected to outcome. As so frequently occurs in drowning accidents, regurgitation of stomach contents complicated the resuscitation efforts. There is no information concerning the victim beyond the fact that he lived in Australia but was heard "talking in another language", and held a scuba diving certificate dated 1969 "not from a recognised organisation". It is perhaps surprising that the available records contain no more information concerning the victim, his health, and swimming/snorkelling, the minimal desirable data. The autopsy showed the presence of diffuse coronary atheroma, but no myocardial infarction. The mitral valve was abnormal with probable insufficiency, and the kidney contained numerous cysts. While the cause of death was drowning there may be significance in the coronary atheroma and mitral valve changes. One witness thought the victim had been wearing special fins but his equipment was not formally described in the depositions.

GROUP SNORKEL DIVE. CURRENT STRONGER AND DIFFERENT DIRECTION TO EXPECTED. SURFACE SAFETY WATCH SLOW TO RECOGNISE DIVERS IN DIFFICULTY. DELAY IN RESPONDING TO OBSERVED NEED FOR ASSISTANCE. POSSIBLY EXPERIENCED SCUBA AND SNORKEL USER. SOME CARDIAC INSUFFICIENCY PROBABLE BUT NO MEDICAL HISTORY.

BH 97/16

The course of events in this case is unlikely to ever

be known. This man was loaned equipment, wet suit, mask, snorkel and weight belt, to enable him to accompany two of his friends when they went to catch fish on a reef, for which they had a license. There is no information concerning either his swimming or snorkelling experience and ability, but as he was able to dive and net some fish at their initial location, depth 3 m (10 ft), he most probably had significant experience. After this they took their boat to their intended location, another dive boat having left the area by this time. He was left in the boat when his two friends commenced their hookah (surface supply) dive and it is probable they did not expect him to remain in the boat to watch their compressor. When one of them returned after about 90 minutes he was surprised to find the victim present neither in the boat nor on the surface nearby. A visual search of the surface was unsuccessful and he then signalled to tell the other diver to surface. They notified the loss of their companion and continued their search until deteriorating weather forced them to stop. The water here was 15 m (50 ft) deep but only 1.5-3 m (5-10 ft) over the nearby bommie. The visibility was good and current minimal. Later official searches were equally unsuccessful, neither body nor any of the equipment ever being found. It is assumed, from the failure of the body to float, that he failed to ditch his weight belt. No reason can be given for him to drown but it is possible he drifted too far from the boat to be able to return, suffered a medical emergency, or possibly suffered a fatal post-hyperventilation blackout. If he was an experienced spear fisherman (there is no evidence of his breath hold diving ability) the last would be a likely scenario.

SOLO BREATH HOLD DIVING WHILE BUDDIES HOOKAH DIVED. ABILITY UNKNOWN. NO ADVERSE WEATHER OR KNOWN HEALTH FACTORS. BODY NEVER RECOVERED.

BH 97/17

On the second day of this live-aboard reef dive trip the victim and his friend were noted to be free diving to 15 m (50 ft) with their spear guns, an indication of their ability. The next day the boat moored at another reef, water depth 25.5 m. While most of the other passengers, snorkel and scuba diver were taken by the boat's tender, the victim and his friend were spear fishing from the stern of the boat, their activities aided by getting one of the passengers to burley the water using pieces of fish. No sharks were seen at this time. A witness described how the victim failed to surface after being underwater for 5 minutes and his float had not moved. Then he realised there was a speargun floating at the end of the line and no sign of the victim, its owner.

The initial was to make a quick surface check using the boat's dinghy. This being unrewarded, the skipper and another person searched for the victim using scuba. They found the spear embedded in coral and surrounded by small sharks attracted by the fish on it. There was nothing in their behaviour to suggest they had attacked anyone. A weight belt was on the line with the spear gun but no trace of the missing man was ever found. It is believed that he must have been negatively buoyant even after ditching his weight belt and that the current had washed the body away over the sandy bottom. He was an experienced spearfisherman, capable of reaching 36 m (120 ft) and was diving to 27 m (90 ft). Possibly he overextended his underwater capability while attempting to free his spear, suffering a post-hyperventilation blackout. Unfortunately ditching his weight belt, an accepted safety measure, failed to save him. He was reportedly a healthy man.

EXPERIENCED SPEARFISHERMAN. SOLO DIVES IN 90 m. CAPABLE OF DIVING TO 36 m. SPEAR FOUND IN CORAL WITH WEIGHT BELT ON LINE. SMALL SHARKS INNOCENTLY PRESENT. BODY NEVER FOUND. PROBABLE POST-HYPERVENTILATION BLACKOUT DROWNING.

Scuba divers

SC 97/1

Although the victim was trained and experienced she was less so than her buddy as she had only dived in summer months while he had continued during the winter. He was taking a course to become an instructor but there was a free day so took the opportunity to join his wife on a wreck dive. She was well trained, having taken deep dive, rescue and rescue courses, but not wreck penetration. The dive plan had to be changed because a large tanker was expected to pass close to the intended location, a wreck, and this would have limited their time there. The nearest suitable alternative at a similar (slightly lesser) depth was a wrecked submarine. Although the divers were given a general briefing there was no specific warning against entering the wreck through its broken hull. The two entered the hull and swam along inside it, meeting at least one other couple similarly occupied. It was close to their ascent time when a silt-out occurred, and despite the buddy's efforts they became separated. The buddy was able to exit the submarine but the victim failed, her body being recovered the next day by police divers using surface supply equipment for safety. She had unfortunately swum into the blind end of the submarine, 35 m from their entry point. There is always a risk of fine silt collecting in any enclosed space underwater and visibility will be treacherously excellent until it is disturbed and a sudden complete loss of visibility occurs, and with it loss of orientation. As direct ascent from such roofed situations is not possible, lack of a line or clear knowledge of the locality can easily result in disoriented swimming until the diver runs out of air.

SCUBA BUDDY PAIR. SEPARATED INSIDE SUBMARINE DURING SILT OUT. DROWNED. DANGERS OF ENTERING ENCLOSED SPACE WITHOUT A LINE.

SC 97/2

During a business visit to Australia, this man and his wife, both trained and apparently experienced divers, chose to join a three day trip to the Barrier Reef. He had made only one dive in the previous 12 months and was taking anti-hypertensive medications (type unknown), but was apparently fit. An instructor was aboard and she gave the passengers a talk during the trip out, mentioning that a personal dive guide could be provided for a small supplement. Although one couple chose to request this option it did not eventuate. The boat was moored about 50 m from the dive site, some bommies. The divers were required to snorkel swim with their guide to where their dive was to commence. The victim and his wife were the last to enter the water but were with the others during this swim. The guide, an instructor, said this couple were checked before the group descended, and when she noted they had not descended with the other 8 divers she surfaced to look for them. She saw them swimming back to the dive boat and that they were being watched from the dive boat.

His wife described how she was surprised when her husband said he was tired and wanted to return to the dive boat as he had not previously mentioned any problem. She swam a fast as possible to avoid delaying her husband. She was about 10 m ahead of him when she looked back and saw he appeared to be drifting. Believing that he might be in need of some assistance she signalled to the boat. There was some delay before she could get a response as the captain thought she was giving an OK signal, then noticed the victim making arm swimming motions and realised he was in trouble. The wife continued her return swim when she saw the tender dinghy start towards her husband, unaware of the seriousness of his condition. When reached, he was unresponsive and very probably dead, but resuscitation efforts were commenced and continued for a time in part to give his wife time to come to terms with his sudden illness. Clinically this was a cardiac death but it was officially recorded as a drowning death because the autopsy did not reveal any significant coronary artery disease, though there was some left ventricle concentric hypertrophy. There is no information available concerning his past health status, and certainly his wife was unaware that he had any heart problem.

EXPERIENCED SCUBA DIVER. SURFACE SNORKEL SWIM. BECAME EXCESSIVELY TIRED. DIED ON RETURN SWIM. BUOYANCY VEST NOT INFLATED. WEIGHT BELT NOT DITCHED. ANTI-HYPERTENSION TREATMENT HISTORY. CARDIAC TYPE DEATH BUT ABSENCE CORONARY ARTERY PATHOLOGY.

SC 97/3

Growing tired of painting and renovating his boat this man, a diving instructor, accepted the suggestion of two of his work mates to go scuba diving from it. Their first dive was successful so they moved to an artificial reef for a second dive. The three men entered the water, two waiting while the third descended. He had strained his knee during the first dive, awakening an old knee problem, so decided to check whether he was fit to make the second dive. After reaching 10 m he decided to abort his attempt. He passed the second buddy, waiting at 7 m to be joined by the victim, during his ascent. The failure of the victim to descend the anchor line puzzled this buddy so he returned to the surface to find out what was causing the delay. To his surprise there was no sign of the missing man, and a descent to the sea bed failed to discover him.

They tried to contact another dive boat which was nearby, but neither radio nor shouting was effective so the first buddy swam across, despite his painful knee, to enlist their assistance. Their searches were unsuccessful and it was only on the third drift dive the next day that his body was seen on the artificial reef. There was a strong current over the reef and this made recovery difficult. The autopsy failed to explain why he drowned as the coronary arteries showed a maximum of 20% narrowing. He was regarded as a careful "do it by the book" workman and diver so it is surprising that he neither inflated his buoyancy vest nor dropped his weight belt. This suggests that death was rapid and unexpected.

TRAINED. SOME EXPERIENCE. DISAPPEARED FROM SURFACE DURING SHORT TIME BUDDIES WERE ABSENT UNDERWATER AWAITING HIS DESCENT. FAILED TO INFLATE BUOYANCY VEST. FAILED TO DITCH WEIGHTS. SUDDEN DEATH. NO CARDIAC CAUSE FOUND. UNEXPLAINED DROWNING

SC 97/4

This was his 17th scuba dive, the 4th on the deep dive course, and the water conditions were good. There were five making this training dive under the care of the instructor and they formed a group of three buddy pairs. They first descended to a ledge at 11m, then continued down to 28 m where they performed tests to demonstrate the presence of nitrogen narcosis before swimming around at 20 m depth until the first one's contents gauge read 100 bar, the arranged indication to start their ascent. It was the victim who reached this cut off first. They ascended as a group to the 5 m deco stop. However the victim continued to ascend, slowly finning till he reached the surface. At no time did he show signs of being in any difficulty.

It was his failure to redescend to the deco stop which led the instructor, and then the others, to surface to find out the reason. They found he had the regulator out of his mouth so the instructor replaced it and inflated his buoyancy vest, then offered to tow him back to the boat. He accepted this offer but made no complaint of having any problem. A short time later the instructor looked back, when he obtained no

response to a question, and saw he was grey and unresponsive. He signalled for the boat to collect them and commenced in-water EAR. Once back on the boat CPR was commenced, but there was no response. There was still 7 bar shown on his contents gauge.

The pre-autopsy X-ray showed the presence of gas in all chambers of the heart, aorta, main limb arteries and in the mediastinum. There was evidence of hypertensive cardiomegaly although the doctor who had performed his pre-training diving medical found his blood pressure only 130/80 supine, 115/95 sitting. It hadbeen 140/90 when checked 10 months previously. He was overweight but was dieting. There was a history of anxiety and he had a script to take half a Xanax (alprazolam) tablet if needed. Lymphocytic thyroiditis was also present. There was clear witness confirmation that he appeared to ascend normally, not rapidly or showing signs of anxiety or urgency.

DEEP DIVE COURSE. HIS 17th SCUBA DIVE. GROUP ASCENT. NO APPARENT PROBLEM TILL HE CONTINUED HIS ASCENT PAST DECO STOP. DELAY BEFORE RAPID DEATH. HYPERTENSIVE CARDIOMEGALY BUT HAD NORMAL RANGE BP. LYMPHOCYTIC THYROIDITIS. MASSIVE CAGE.

Surface supplied diving

H 97/1

Although, according to his father, he never successfully completed any of the courses he attended, this man is reported to have held a commercial diver license, owned a dive store and trained others. The truth or otherwise of these comments is not documented in the official records. He was reportedly always careful with the state of his equipment, but when it was examined following his death it was found to have several significant faults, water in the compressor's reservoir, the demand valve allowed in water, the hose couplings were not secured by tape and easily disengaged, and the air intake was unsecured and lacked an air filter. There were three friends in the boat, one being a commercial diver and the other an untrained diver who was experienced by diving with the victim. The former regarded the victim as being reasonably experienced. They were, unsuccessfully, hunting for crayfish among thick kelp, two being down at a time. The buddy became tired and returned to the boat and while he was removing his equipment the hose came off the compressor. He jumped back into the water to retrieve the hose and pulled it and the victim, who had now surfaced, back to the boat where the third diver reattached the hose. The victim was shouting for help and thrashing his arms about at this time, but submerged again after the hose was reattached, which surprised the other two. The thick kelp made it difficult to pull him back to the surface and get him into the boat. Their CPR efforts were unsuccessful.

The pre-autopsy X-ray showed the presence of air within the heart, clear evidence of CAGE. It is thought he probably failed to exhale adequately during his no-air ascent and the thick kelp would have restricted his free ascent to the surface. The comments of the state of his equipment showed that it was in an unsafe condition despite his reputation for safety consciousness. When the equipment was officially examined it was noticed that the bail out bottle was not connected, but it is not known whether this was due to the victim releasing it in his attempt to activate it, occurred during his retrieval, or occurred during its transport and storage before it was checked. Although there was a family history of heart trouble and he was overweight at 108.2 kg (17 stone or 238 lb) there was no evidence of hypertension or coronary artery disease.

HOSE SUPPLY DIVING IN KELP. EXPERIENCED BUT TRAINING UNCERTAIN. HOSE SEPARATED FROM COMPRESSOR. DELAYED ASCENT. ADVERSE COMMENTS CONCERNING EQUIPMENT. BUDDY ALSO EXPERIENCED BUT UNTRAINED. DELAYED ONSET SYMPTOMS OF CAGE.

H 97/2

The importance of never using faulty equipment, and of ensuring that it is competently maintained, was tragically demonstrated in this incident. One of the three friends collected cowrie shells for his personal collection, strictly not for sale as he was not licensed. His two friends helped him and the previous day had found that the compressor they used was unable to maintain an adequate air supply for two at 36 m depth. They had replaced the reed valve on the middle cylinder and expected this would be sufficient. However they soon found it would still only supply one diver at the desired depth.

It was the victim's turn to dive. Although he did not like it he was wearing a bail-out bottle on his harness. After he had been underwater a short time the two who remained in the boat noticed the pressure was falling and increased the engine to full throttle. This improved the pressure for a short time, then it again began to fall and this became increasingly rapid. They decided to warn the diver to ascend by two pulls on the hose. They are uncertain whether there was any response. They began to pull up the hose slack, taking in 10 m and indicating the diver was ascending, then it began to run out and it felt as if there was a dead weight at the end of the hose. This led them to decide to pull their friend up using the hose, which to their surprise now ran under the boat. When they had pulled him to 4-5 metres below the surface they could see his regulator was free flowing and one of them dived down to find whether he wished to make a deco stop. He found him hanging limp, the regulator hanging free. They pulled him into the boat and performed CPR but this was unavailing. As there was

no response to their radio calls for assistance, they returned to shore and transported the body to the nearest hospital.

The pre-autopsy X-ray showed the presence of not only a left pneumothorax and surgical emphysema but air in the heart. While these findings may have been modified by their CPR efforts, this was clearly a severe case of pulmonary barotrauma with CAGE. He was reputedly a calm, conscientious, safety conscious and experienced diver, very probably scuba trained, who had frequently used hookah apparatus. However these three divers had apparently dived using a compressor they knew was working below its correct efficiency. The report on the examination of the compressor found numerous faults. The reed valve in the central cylinder had snapped (they heard this occur but did not realise the significance of the sound), there was a major leak from the central metal filter of the compressor, all the delivery fittings had leaks, and the intake hose was plastic and had melted and fused. This last problem could have led to the creation of carbon monoxide. He was reported to enjoy the feeling of nitrogen narcosis, a somewhat unsafe addiction.

EXPERIENCED HOOKAH DIVER. DEEP DIVE FOR COWRIE SHELLS. MALFUNCTIONING COMPRESSOR MINIMALLY REPAIRED. LOSS OF AIR PRESSURE THROUGH COMPRESSOR LEAKS. ASCENDED ABOUT 10 m THEN APPARENTLY LOST CONSCIOUSNESS. LEFT PNEUMOTHORAX. AIR IN HEART. SURGICAL EMPHYSEMA. CAGE.

Discussion

It is highly probable that two factors explain the rarity of reports of snorkel user/breath-hold other than in Queensland. First there is the factor that the Great Barrier Reef is known world wide and attracts large numbers of visitors from overseas and other States, many of mature age and with little or no previous experience of snorkel use, which weights the numbers who are at risk. Second, this is so important to the local economy that Government and press show particular interest in such incidents as occur. This media and Government interest does not exist in other States and it is therefore probable that such fatalities as occur fail to receive more than local public notice. It is noted that only one of the cases recorded here occurred outside Queensland and that was an experienced diver who surfaced in the path of two fishing boats and was hit by the propeller of one. Given the bias to age in Reef visitors it is perhaps not surprising that a cardiac factor has been considered the critical element in ten (10) of the fatalities. In one a post-hyperventilation blackout, followed by drowning, is believed to have occurred. However there is no clear explanation for three cases, but it is possible that the critical factor was unfamiliarity with use of a snorkel. Certainly water conditions, and even water depth, do not appear to have been factors. It is noticeable how silently death can come, the victim giving no sign of experiencing any problem to persons nearby. In one case (BH 98/16) the body was never recovered and insufficient is known concerning the victim's abilities to suggest a possible cause for his death, while in the other case where the body wan never recovered (BH97/17) the victim's health and ability were documented.

There were four (4) identified scuba diving deaths. The tragic death due to a silt-out while exploring inside a sunken and broken submarine underlines the warning that any enclosed space is likely to contain fine silt which changes temptingly crystal clear water into impenetrable fog in an instant once disturbed. Cases SC 97/2 and 97/3 are difficult to explain other than supposing a sudden cardiac event of which there was no hard evidence at autopsy. Case SC 97/4 illustrates the maxim "Man proposes, God disposes" as this was a normal ascent in a group during which, apparently, the diver suffered a massive air embolism and pulmonary barotrauma. While it possible to invoke stress due to his relative inexperience, and nitrogen narcosis at depth causing him to omit correct breathing, nothing untoward was observed in his behaviour by his more experienced companions.

Surface supply is, in the well-ordered world, a marvellous way to dive as there is an unlimited duration of air supply. Unfortunately in the real world surface supply provided by a compressor (hookah) can cease with minimal warning, with tragic results for an unprepared diver. In the first case the unsatisfactory functioning of the compressor was warning of possible trouble, though naturally none expected the sudden critical failure of the air compressor. The thick kelp complicated the recovery of the victim. Whether or not the victim attempted to use his bail-out bottle cannot now be known but he certainly suffered air embolism, though this was not immediately fatal as he was able to shout and thrash about at the surface before sinking again. A similar catastrophic loss of air occurred in the second case and here there was a depth factor and the embolism appears to have been fatal well before he reached the surface.

To summarise, inexperienced snorkel users appear be at some risk even in shallow, calm water, and there is a significant risk of critical cardiac events in the older group of users. The cardiac risk should also be remembered in relation to scuba divers. The danger of entering areas from which direct ascent to the surface is impossible is very real if fine silt has accumulated, so should always be remembered. Surface supply users must be aware of their dependence for survival on a guaranteed air supply and hose line. Acceptance of less than this standard can be fatal.

Acknowledgments

This investigation would not be possible without the understanding and support of the Law, Justice or Attorney

General's Department in each State, the Coroners, and police when they are approached for assistance.

PROJECT STICKYBEAK

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

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 P.O. Box 120, Narrabeen, New South Wales 2101, Australia. Fax + 61-(0)2-9970-6004

ARE SOME JELLYFISH TOXINS HEAT LABILE?

Geoff Taylor

Key Words

Injury, marine animals, toxins, treatment.

Abstract

The author reports four episodes of stings from the Jellyfish *Tamoya gigantua*, two of which are his personal experiences. On the second occasion the sting was successfully treated with local heat. This raises the question as to whether other jellyfish toxins are heat labile and could be treated in the same way.

Introduction

Jellyfish stings of varying severity are a very common problem for Australians pursuing water sports around our coasts. They vary from the relatively innocuous sting of the Jimble (*Carybdea rastoni*) to the more serious Irukanj syndrome and Box Jellyfish stingers. Local first aid measures that have been recommended for the less severe stings include the use of vinegar, aluminium sulphate (Stingose), lignocaine jelly, papain meat tenderiser and ice.[1,2]

Tamoya gigantua is a little known jellyfish that frequents the tropical waters of northern Australia. It is a large box-jellyfish with an elongated box structure and has only four, very short, thick tentacles at each corner. There is commonly a small fish (species unknown) that resides in the bell of the jellyfish. In appearance it superficially resembles a large innocuous comb jelly and does not look like a stinging species. However, the whole body of the animal is covered in nematocysts that pack a powerful punch and can penetrate protective clothing.

Case reports

The first case was a bather who had been swimming near the Navy Jetty at North-West Cape, near Exmouth (latitude 21° S) and suffered a powerful sting. A local diver was despatched to investigate any species likely to have caused this injury. He returned with a huge specimen of *Tamoya* whose bell was 22 cm long. No one was game to experiment with this giant and we remained unsure if it was the culprit.

It was several years later that a diving companion recounted his own experience of being stung in 1987 by the same species, on the hand, while diving at Point Cloates on the Ningaloo Reef. He had suffered intense local pain, which spread to his axilla. He felt tightness in the chest making him short of breath. The pain lasted for several hours causing considerable fear and distress. His diving trip was curtailed and the dive boat returned many miles to base-camp.

The next two cases are my own experiences of being stung on two occasions and the successful use of heat to treat the pain of envenomation.

The first occasion occurred on Ningaloo reef in 1994. While swimming in deep water awaiting pick-up by a boat, my left knee struck a sizeable *Tamoya*. The sting penetrated through a lycra bodysuit, causing instantaneous severe burning pain. The pain soon spread to regional lymph glands in the groin, but there were no systemic symptoms. The intense pain lasted for about two hours, and then slowly subsided over the ensuing three hours.

The second event occurred in the same locality, a year later. On this occasion, while snorkelling, my head struck the *Tamoya*, the stingers penetrating my hair (which is surprisingly thick), with extensive stinging over the scalp. At the same time I lifted my hand in a reflex action to fend off the "attacker", and was stung on the back of the hand.

On this occasion it was decided, as an experiment, to try treating the sting with heat. My hand was immersed in a bowl of hot water as hot as I could stand. This brought almost immediate relief of the pain, but initially the pain recurred after removal from the heat. After 20 minutes the

effect of the heat treatment was persisting. However, the pain in the scalp had spread to the neck probably though lymphatic spread. There was an intense burning, and a feeling as if my head was in a vice.

Hot towels were tried and a hot shower, but in the end I was subjected to lying prone on a bench with my head in a bowl of hot water. The relief was very rapid and after 20 minutes the pain had lessened to such a degree that treatment was ceased. Within an hour of being stung I was virtually pain free, and able to resume diving.

Discussion

The toxins of many marine species are known to be heat labile. These are principally members of the Scorpion Fish and Stingray families. Toxic spine injuries from these species are successfully treated with hot water.

This report suggests that the toxin of the jellyfish *Tamoya* is heat labile and able to be treated with local heat. This raises the question as to whether other species of jellyfish sting can be treated in this way.

The author is now resident in Busselton, Western Australia, on Geographe Bay. This area is well known for its summer plague of "stingers"; the principal species is thought to be the "Jimble" *Carybdea rastoni.* The severity of the sting received by subjects is very variable and some individuals seem to have a hypersensitivity to these stings, with the development of large wheals that take several days to resolve. Others only experience a transient stinging sensation and mild erythema.

Some hypersensitive individuals, who have been told of the benefits of immediate heat treatment, have reported to the author that heat treatment after being stung resulted in considerable improvement of their symptoms.

The Tamoya jellyfish is not a life-threatening species and stings with this species are rare. However, my own experience and the reported improvement in symptoms in sting-sensitive individuals who have used heat (hot water) treatment after being stung by unknown jellyfishes in Geographe Bay raises the question that perhaps the pain of more jellyfish stings might be alleviated by immediate heat treatment.

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VALIDATION OF THE RNZN SYSTEM FOR SCORING SEVERITY AND MEASURING RECOVERY IN DECOMPRESSION ILLNESS

Tony Holley

Key Words

Decompression illness, risk, treatment sequelae.

Abstract

A retrospective study of 100 divers with a provisional diagnosis of decompression illness (DCI) admitted to the RNZN Slark Hyperbaric Unit from June 1995 to February 1997 inclusive, using the Royal New Zealand Navy (RNZN) scoring system for assessing the severity of DCI and recovery with treatment,[1] was carried out. Only 79 of the divers fulfilled the conditions of entry into the study, 21 being excluded because of doubtful diagnosis, inadequate case notes or a diagnosis of cerebral arterial gas embolism (CAGE). These latter, because of the Unit's protocols, were kept horizontal until under pressure so could not have their standing and walking ability assessed.

The study showed that 59 out of 66 (89.4%) divers with a score of 25 or less (≤25) on admission had a symptom free recovery after treatment, or a sequelae rate of 10.6% (7 of 66). Of the 13 divers with an admission score of more than 25 (>25) only 3 were symptom free after treatment (23.1%) while 10 (76.9%) were left with sequelae.

The RNZN DCI scoring system has good prognostic power. The admission severity score correlates linearly with severity, as indicated by the number of treatments required to achieve maximum recovery, confirming that it is a useful index of severity when assessed at the time of presentation for treatment.

Introduction

There is a lack of information on prognostic factors in DCI, in contrast to many other conditions, such as the Critically Ill,[2] Head Injury,[3] Meningococcal Septicaemia,[4] Multiple Trauma[5,6] and Acute Pancreatitis,[7] in which epidemiological and clinical studies have elucidated

prognostic indicators that have proved useful in the classification and treatment of the condition.

Although there have been a number of scoring systems devised for decompression illness[8-12] none of these meets the requirements of providing:

1 a universally applicable system for all forms of DCI;
2 a numerical severity index at presentation;
3 a numerical index of progress and recovery;
4 a methodology for comparing different therapies in different diver populations.

A scoring system for decompression illness (DCI) which aims to quantify relative severity of disease at presentation, and relative recovery after treatment was established by Mitchell et al. at the Royal New Zealand Navy (RNZN) Slark Hyperbaric Unit.[1] The prognostic validity of this system was assessed by this study.

The RNZN scoring system is complex in its derivation, but simple and convenient to employ clinically.[1] Numerical analogue scales were derived for each symptom or sign. An "importance" conversion factor for each potential manifestation was derived by ranking each symptom or sign on scales for four parameters (specificity for DCI, natural history of that manifestation if untreated, potential for incapacity and a co-dependence compensation). A second conversion factor accounts for the time course of any particular disease manifestation, and was generated by arbitrarily allocating a numerical weighting to the descriptive terms used by Francis and Smith (static, remitting, relapsing and progressive).[13] Assessment of the clinical course before and after treatment, allows calculation of a "severity" index and a "recovery" index for any patient.[1] The RNZN scoring system will be useful, as both a research tool in comparing therapeutic modalities and for the assessment of disease severity and the effects of treatment in the individual diver.

Scoring systems are generally constructed by identifying (either by clinical consensus or statistical analysis) variables which are best related to outcome. Weights are then attributed to those variables to generate a score, as was the case for the RNZN system. Before adopting a prediction rule or scoring system, clinicians must evaluate its applicability to their patients.[14]

In this study the RNZN DCI scoring system was evaluated by assessing the prognostic value of the severity score at admission in a large, heterogeneous population of injured divers.

Method

The RNZN decompression illness severity scoring system was validated by reviewing the case notes for all divers presenting with DCI treated at the RNZN Slark Hyperbaric Unit from June 1995 to February 1997 inclusive.

This retrospective review yielded 100 cases. Twenty one were excluded from the study. Exclusion criteria were:

1 equivocal diagnosis;
2 inadequate documentation in the clinical notes to allow for reliable severity score calculation;
3 patients treated for cerebral arterial gas embolism, as it is unit policy to keep these patients in the supine position until recompression therapy has been commenced.

The study population had a diverse spectrum of disease including neurological, musculoskeletal, constitutional and cutaneous decompression illness.

Admission severity scores were retrospectively calculated for those patients meeting the inclusion criteria. Severity scores were also calculated from the clinical records at discharge. Each patient's recovery index was calculated by subtracting the discharge score from the admission score. Admission scores were also correlated with the number of once daily hyperbaric treatments required to achieve maximal recovery (defined as either full recovery or failure to record sustained improvement over two consecutive days). The predictive values of scores >25 and ≤25 at admission were compared for incomplete recovery at discharge from hospital to give a negative (sequelae present) prediction rate and a positive (complete cure) prediction rate. The prognostic value of the new scoring system was determined by calculating the positive and negative predictive values for a score of less than, equal to or greater than twenty five.

Demographic data for all 79 patients included were recorded (Table 1). There were 71 males and 8 females, the mean age was 33.5 (± 9.1) years. For all cases the time from the last dive to presentation was established. There was a mean delay of 53 (±71) hours and objective signs were detected in 61 patients (77%). The mean number of treatments was 5.2 (± 4.2). In 62 patients (78.5%) full recovery was documented. Seventeen patients (21.5%) were discharged with sequelae.

TABLE 1

STUDY POPULATION

Diver total	79
Males	71
Females	8
Mean age	33.5 (± 9.1) years
Objective signs at presentation	61 (77%)
Complete recovery	62 (78%)
Mean delay to presentation	53 (±73) hours
Hyperbaric treatments per diver (mean)	5.2 (±4.2)

Microsoft Excel software was used to establish the distribution of the severity scores for the study population. For each score, the number of patients demonstrating recovery or sequelae were compared. Using a linear regression analysis model, the relationship between the initial severity score and the number of treatments required to achieve full or maximal resolution for individual patients was established. At the RNZN Slark Hyperbaric Unit patients are treated on a daily basis until full resolution or until, despite two further treatments, a clinical plateau is achieved. In the absence of a single marker for the severity of decompression illness, the number of treatments required to achieve "best" resolution provides a useful retrospective indicator of disease severity.

Results

The study showed that those with a score of 25 or less (≤25) on admission had a symptom free recovery rate after treatment of 59 out of 66 or 89.4%, or a sequelae rate of 10.6% (7 of 66) (Table 2). Of the 13 divers with an admission score of more than 25 (>25) only 3 were symptom free after treatment (23.1%) while 10 (76.9%) were left with sequelae. These results show that for an admission score of ≤25 the likelihood of positive result (complete recovery) is 89% and for an admission score of >25 the likelihood of a negative result (incomplete recovery) is 77%.

TABLE 2

PROGNOSTIC VALUE

Score	Sequelae	Recovery	Total
>25	10	3	13
<25	7	59	66
Totals	**17**	**62**	**79**

Positive predictive value = 77%
Negative predictive value = 89%

There was a strong linear correlation between admission severity score and number of treatments (multiple r = 0.80; r^2 = 0.64). Table 3 (page 78) shows the discharge score ranges of the divers left with sequelae, their individual discharge scores and their symptoms and signs. It is clear that those with a discharge score of 25 or less were less handicapped than those with scores of 36 or over.

Discussion

This study of a large population of divers who presented with heterogeneous manifestations of DCI has tested RNZN DCI severity scoring system and demonstrated that it can be used to follow the progress of patients in response to hyperbaric treatment and to predict the likelihood of permanent sequelae after treatment to "no further improvement".

Other authors have proposed gravity or severity scoring models, but none of the systems has been applicable to a wide range of clinical presentations.[8-12]

Ball et al.[10] produced a model which was intended for use specifically in neurological DCI and included historical, therapeutic and clinical parameters. The authors stated "this gravity index is in no way intended for application to individual cases". In addition to exclusion of the common musculo-skeletal DCI, this system is insensitive to those divers with primarily dorsal column spinal lesions and those divers who lack objective neurological findings. This alone would preclude their system's use in excess of 50% of patients presenting to Australasian hyperbaric facilities.[15]

Boussuges et al.[11] devised a scoring system which is useful for "assessing the gravity of a population with a view to comparing the efficiency of different therapeutic protocols". However, this system again effectively disregards a large subgroup of patients with neurological symptomatology in the absence of objective neurological findings.

Valuable work by Kellher et al.[12] produced a system capable of predicting the probability of incomplete resolution after the first recompression intervention. The authors, however, excluded cognitive disorders, abnormalities of special senses and sphincter dysfunction, claiming they were infrequent and hence unlikely to facilitate development of a model. In Australasian experience these presentations are not infrequent.

The RNZN DCI scoring model, subjected to validation in this study, encompasses a wide range of potential presentations. The model is highly inclusive and it is simple and time efficient to implement.

Demographic data obtained from the study population revealed a mean age of 33.5 (±9.1) years and 9:1 male to female ratio. The marked male predominance and age distribution is common in Australasian facilities treating recreational divers.[15-18] However, this study has a higher male predominance than most other series, where the male predominance is usually in the order of 70%. There is no obvious explanation for the male bias in this study sample.

The mean delay to presentation was 53 (±73) hours, which is significant in that it reflects the inclusion of very mild or subtle disease. One would expect that there would be very little delay in presentation in the presence of severe

TABLE 3

Scores and Sequelae at Discharge

Score Range	Number of divers	Individual Scores	Sequelae
6-10	2	9	Musculoskeletal pain
		10	Musculoskeletal pain
11-15	1	11	Paraesthesiae hand
16-20	1	20	Mild cognitive impairment
21-25	3	21	Objective sensory deficit foot
		23	Diffuse musculoskeletal pain
		25	Paraesthesiae arm
26-30	2	27	Mild facial paraesthesiae
		29	Tinnitus right ear
36-40	1	37	Bilateral lower limb weakness (able to ambulate without assistance)
41-45	1	45	Bilateral thigh paraesthesiae
46-50	1	48	Mild cognitive impairment
51-55	2	51	Mild cognitive impairment, musculoskeletal pain
		54	Lower limb weakness (unable to walk without assistance) Objective sensory deficit in lower limbs
56-60	2	56	Gait disturbance (ataxic), musculoskeletal pain, Paraesthesiae
		59	Labile affect, moderate cognitive disturbance
66-70	1	71	Paraplegia, bladder dysfunction, lower limb sensory loss

symptomatology, and what delay did occur would be a function of transportation times. Unfortunately this is not always so. It would seem likely that those with mild disease, or that which was perceived to be insignificant, might well delay their presentation.

The distribution of the injured diver population according to the admission severity index score, and post treatment sequelae (Figure 1), provides a useful tool. The significant difference in the severity score between the divers with sequelae and those who recovered completely is the first step toward validation.

The prognostic value of the severity score above 25 was established by calculating the positive and negative predictive values. Ten (76.9%) of the 13 divers with a score above 25 developed sequelae (Table 2). Conversely 59 (89.4%,) of the 66 divers with a score less than or equal to 25, did not develop sequelae. Analysis of the prognostic value of scores higher than 25, therefore confirms the validity of this severity index. A negative predictive value for a score of equal to or less than 25 is useful in advising patients as to the probability of full recovery.

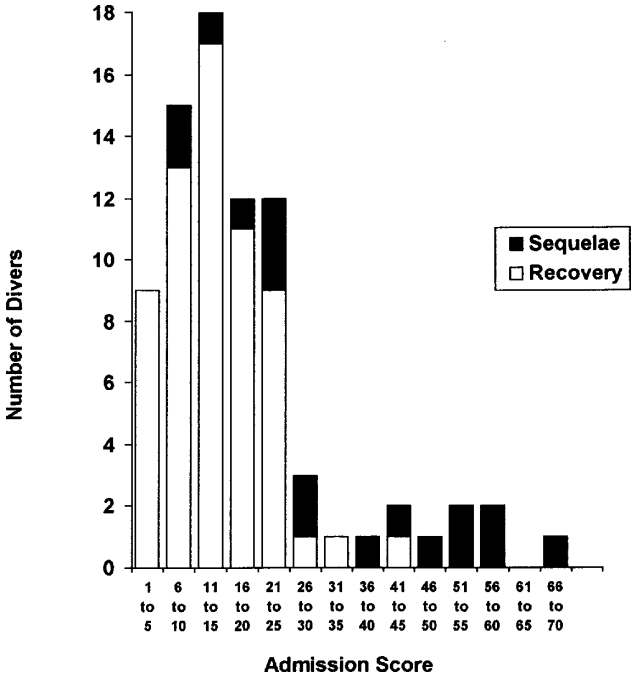

Figure 1. Distribution of admission scores.

Admission score versus number of treatments (Figure 2) provides a linear relationship, with a correlation coefficient of 0.80. In the absence of a single marker for the severity of DCI, the number of treatments received by the patients has been utilised to best reflect severity. The RNZN Slark Hyperbaric Unit treats all injured divers until full resolution, or until a clinical plateau is achieved (as determined by two further treatments failing to demonstrate

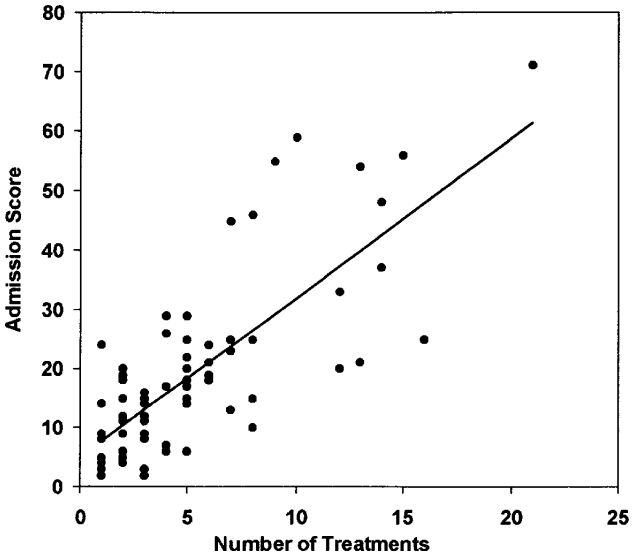

Figure 2. Admission score verus treatment number ($r=0.80$, $r^2=0.64$)

any improvement). The Undersea and Hyperbaric Medical Society Therapy Committee Report[19] provides upper threshold limits for therapy in DCI and indicates therapy should be continued until "improvement plateaus or 14 days". No patients who did not improve with treatment received more than two such "failed" treatments. Patients in this study received a mean number of treatments of 5.2 (±4.2), with three patients receiving in excess of 14 days therapy (receiving 15, 16 and 20 treatments respectively). These three patients were treated beyond the 14 day recommendation as they continued to improve. If these three patients are excluded from the study, the correlation coefficient is 0.74 . The implication of the linear relationship between admission severity score and number of treatments, is that for an individual patient for whom a severity score is calculated, the number of treatments required may be reliably estimated. This information will not only be useful for the physician's treatment planning, but also for the divers and their families, who are often resident in different geographical locations to the hyperbaric facility.

The purpose of the RNZN DCI scoring system is to assign a numerical index of severity, rather than attempt to describe the exact character of any sequelae.[1] For completeness, the sequelae experienced by the study population have been included (Table 3). Only one diver, of those individuals presenting with an admission severity score ≤25, was discharged with potentially disabling sequelae (headaches and mild cognitive impairment). All patients with an admission severity score of 48 and above were discharged with potentially severe disabilities.

Clarification of the prognosis of DCI treated with standard therapies is important for several reasons. Firstly, education of the patient during treatment is important. Some divers have the misinformed belief that DCI is easily treatable and always cured with recompression therapy and, thus, expect complete resolution following treatment.[12] Secondly, the estimated likely number of treatments until full recovery or plateau will be useful to the patients and their families. Thirdly, identification of patient sub-groups with a poor response to standard therapy could be useful in the initiation of early adjuvant therapies.

Finally, a reliable classification of the prognosis allows for comparison of injured diver populations. The Slark Hyperbaric Unit at the RNZN Hospital has initiated a randomised, prospective, controlled, double blind trial of lidocaine as an adjuvant to recompression therapy in the treatment of DCI. For the purposes of this trial, a scoring system, which provides an effective and quantitative method of tracking progress and assessing recovery is required.

Boussuges et al.[11] state that the use of clinical criteria alone in a severity score could limit its reproducibility and hence suggest the inclusion of objective criteria such as haematocrit (which they believe to correlate with prognosis in decompression illness). The RNZN DCI severity index includes a wide range of clinical parameters, but also clearly defines how each should be applied with the intention of retaining reproducibility.

While the results of this validation study suggest the severity index is a good predictor of improvement with treatment, the likelihood of sequelae and a useful tool for research, several caveats must be recognised before attempting to generalise these results.

The clinical-descriptive classification of decompression illness[13] refers to the full spectrum of disease that results from decompression and the consequent lowering of ambient pressure. This descriptive classification therefore, includes Cerebral Arterial Gas Embolism (CAGE). At the RNZN Hyperbaric facility, those patients suspected of recent CAGE are not tested for gait or balance, for fear of posturally induced arterial gas embolism, but are maintained in the supine posture until under pressure in hyperbaric therapy. The RNZN scoring system has, therefore, not been applied to this sub-group of patients and cannot be considered a useful entity in the assessment of CAGE on admission.

The large proportion of cases (21%) that were excluded from analysis because of incomplete notes, equivocal diagnoses or inadequate clinical examination and CAGE, increases the probability that the conclusions drawn from the sample population are biased. This would be the case if the study variables in the missing records should differ from those in the study population. It is impossible to use missing records and it is normal practice to base conclusions on those records which are complete. The solution is better recording by medical staff of all aspects of treatment so that fewer patients have to be excluded.

As symptoms, signs and recovery were recorded together in the medical records, it was not possible to blind the severity scoring process. Furthermore, all scoring and data collection were performed by the same researcher, which could possibly bias the results. This is an unavoidable problem with retrospective research.

Conclusion

Validation of the RNZN scoring system, using a retrospective review of 100 cases, has demonstrated that it has good prognostic capability and is useful for research. The RNZN index now ready to be validated in a prospective, multicentre study.

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Acknowledgments

Special thanks to Dr Des Gorman, Associate Professor of Medicine at the School of Medicine, University of Auckland, and Visiting Consultant in Diving and Hyperbaric Medicine at the Slark Hyperbaric Unit and Dr Simon Mitchell, (formerly) Director of the Slark Hyperbaric Unit at the RNZN Hospital for their continual help and support with this diploma project.

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The above paper is an edited version of the thesis presented by Dr Holley for the SPUMS Diploma in Diving and Hyperbaric Medicine which was awarded to Dr Holley in May 2000.

THE WORLD AS IT IS

CERTIFICATION IN DIVING AND HYPERBARIC MEDICINE IN AMERICA AND AUSTRALIA

John Knight

Key Words
Qualifications.

The 2000 May/June issue of PRESSURE contains two articles about Certification in Undersea and Hyperbaric Medicine in the United States of America. Although the SPUMS Diploma of Diving and Hyperbaric Medicine (DDHM) was introduced in 1974, and has become the de facto qualification in Diving and Hyperbaric Medicine in Australasia, Diving and Hyperbaric Medicine has not been accepted as a speciality, or even a sub-speciality, in Australasia.

The United States has, thanks to many years of negotiation between the Undersea and Hyperbaric Medical Society (UHMS) and the American Board of Medical Specialties (ABMS), become the first English speaking country to accept Diving and Hyperbaric Medicine as a speciality, albeit a sub-speciality (labelled Undersea and Hyperbaric Medicine) held by someone who is already Board Certified in some other speciality. This new sub-specialty certification is now a recognised element in the American Medical Association's system of certification. Diving and Hyperbaric Medicine has come of age in America.

The first examination for certification in Undersea Medicine was administered by the American Board of Preventive Medicine (ABPM) in November 1992. Last year the American Board of Medical Specialties (ABMS) agreed to change the sub-speciality's name to Undersea and Hyperbaric Medicine to reflect the expansion of practice during the past decade to include regular use of hyperbaric oxygen therapy for a variety of disorders. The first examination for this qualification was held in November 1999. Fifty candidates passed the exam, including the President of UHMS, Dr Caroline Fife, and two past Guest Speakers at SPUMS Annual Scientific Meetings, Drs Fred Bove and Richard Moon.

This November an examination, sponsored by the American Board of Emergency Medicine (ABEM) and ABPM and approved by ABMS, for certification in Undersea and Hyperbaric Medicine will be held. The date for applying to sit is now past but UHMS will be running a Refresher Course in Hyperbaric and Undersea Medicine in Bethesda, Maryland on 23 September. Everyone who took the Refresher Course last year in San Antonio passed the certification exam. Those wishing to attend the 2000 course should notify the UHMS office before 2000/7/1. The examination is open to all physicians boarded in any primary medical specialty, viz. medicine, surgery, emergency medicine, etc. who have completed one of two eligibility pathways either an Undersea and Hyperbaric Medicine Fellowship or Undersea and Hyperbaric Medicine Training Plus Practice.

Eligibility criteria are available from the two sponsoring Boards at <http://www.abem.org> and <http://www.abprevmed.org>.

In Australasia the Special Interest Group (SIG) in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists (ANZCA) is formulating a training scheme and syllabus to extend Diving and Hyperbaric Medicine education beyond the requirements of the SPUMS diploma. It is investigating asking the ANZCA to set up a certificate of training and competence in diving and hyperbaric medicine, which will be a higher qualification in the these subjects. This path has been followed by the SIG in pain medicine, which has graduated to being the Faculty of Pain Medicine and is open to non-anaesthetists who are involved in pain relief.

As can be seen from the letter from the Chairman of the SIG on page 88, there have been some teething problems. Anyone interested in the Diving and Hyperbaric Medicine SIG, which has been formed to provide a forum, beyond the Annual Scientific Meetings of SPUMS, for discussion about diving and hyperbaric medicine, and to educate other members of the medical profession, especially anaesthetists, emergency physicians, intensive care physicians and those involved in all branches of surgery where hyperbaric oxygen can be expected to improve patient treatment and outcome, should contact to Ms Helen Morris at the ANZCA for further information and to register their interest.

The address of the Australian and New Zealand College of Anaesthetists is 630 St Kilda Road, Melbourne, Victoria 3004, Australia.

Ms Morris' e-mail address is <helen@anzca.edu.au>, telephone +61-(0)3-9510-6299 and fax +61-(0)3-9510-6786.

DIVING DOCTOR'S DIARY

WAS IT DECOMPRESSION ILLNESS ? PROBLEMS WITH DIVING AND DOCTORS

Carl Edmonds

Key Words

Case history, decompression illness, medical conditions and problems.

Case history

A 45-year-old slightly obese, experienced female diver was treated for two episodes of acute decompression illness, neurologically static, which responded to repeated hyperbaric treatments.

This enthusiastic lady has been diving for five years, mainly in warm waters and at moderate depths, rarely exceeding 18 m. She employed a dive computer, but never entered decompression. Two years ago she undertook her first diving holiday, on a live-board, undertaking multi-day and repetitive dives.

The first episode of decompression sickness was noted after 3 days on board. It occurred a few hours after the diving, and comprised paraesthesia over the first, second and third fingers and associated joints of the right hand, together with some pain and discomfort, weakness and clumsiness affecting the hand. The left hand was also affected to a slight degree, mainly involving the thumb and first metacarpophalangeal joint.

Some relief was noted during further dives, but it was not until the diving had been completed, and she had been given three oxygen recompression therapy sessions that she really improved. Even then, the improvement was gradual.

Single dives carried out over the next year were uneventful, but then a return to live-board diving caused a recurrence of almost identical symptoms and progress. The only addition to the sequence of events was a slight worsening of symptoms during the altitude exposure, flying back to Sydney.

The reason for her consultation was to determine whether or not her diving activities should be altered.

Diagnosis

As so many of the current diving physicians are taught, any diver with symptoms following a dive should be treated as if they have acute decompression illness (the fashionable terminology). Indeed, some of the characteristics of decompression sickness are present in this case i.e. excessive diving exposure, relief with re-immersion, improvement with recompression therapy, aggravation by altitude exposure. Mild obesity and age were predisposing factors.

If, instead of employing the venerable diving principles espoused by our predecessors, including a "trial of pressure", one employs the more traditional Oslerian medical approach to the case, a different pattern emerges.

She gave a history of very mild hypertension, osteoarthritis and the development of peripheral oedema, over the previous two years, fully investigated and not shown to have any cardiac, renal or metabolic aetiology. Her oedema had previously been very well controlled on Moduretic (amiloride 5 mg and hydrochlorothiazide 50 mg).

On examination it was evident that some of the symptoms could be replicated by pressure over the carpal tunnel, and the sequence of events then became clear.

As suggested by her physician and her diving instructor, she had stopped taking the diuretic during the period of intense diving on the live-aboard. This had resulted in a gradual redevelopment of her peripheral oedema, and the production of a typical carpal tunnel syndrome, worse on the right side. Possibly immersion assisted in reduction of the peripheral oedema, also reducing the symptoms, at least for a few hours, then the oedema redeveloped. The exacerbation of her symptoms during flight might be related to the aggravation of the ischaemic affects of the carpal tunnel pathology.

Once she had finished diving (and also during the period of recompression therapies) she resumed the diuretic regime and thereby relieved her symptoms.

Prognosis

A repeat of the investigation for the cause of the oedema was unrewarding, it now being classified as idiopathic (not that uncommon in middle-aged females). She now continues her diuretic regime during her diving activities, and has had no recurrence of the "bends". As her diving was, even previously, extremely conservative and within no-decompression limits, it was not thought necessary to modify this.

Background

The carpal tunnel syndrome is an entrapment neuropathy of the median nerve at the wrist. Paget first

described this in 1854 in a patient who sustained a fracture of the distal radius. It has recently been reviewed by Slater.[1] The carpal tunnel is enclosed by the bones of the wrist, roofed over by the flexor retinaculum fascia. It contains nine tendons and the median nerve which lies most superficially, immediately under the ligament. The basic physiology is a reduction in the epineural blood flow which occurs with compression of 20-30 mmHg.

It may be aggravated by arthritis affecting the wrist joints, positioning of the hand and wrist, oedema of the enclosed tissues, or anything else which compromises the space available for the median nerve in the tunnel. Thus local causes, such as radius fracture, blunt trauma with haemorrhage and swelling, various tumours, systemic illnesses, metabolic diseases, overuse syndromes and aberrant anatomical structures have been incriminated.

Support for the diagnosis is based on involvement of the median nerve distribution and referral area (symptoms related to the first three fingers, thenar eminence, wrist, arm and even up to the shoulder). Pressure over the compressed nerve area, either directly or by reducing the venous return, may provoke the symptoms (Tinel's sign).

Discussion

This case highlighted three dubious diving medical dictums.

1 *Symptoms following diving should be classified as decompression sickness*. This may have been so during the early days of diving, when most divers were young fit males, usually in the Navy and often employing less reliable decompression regimes, and with a diving physician and recompression chamber nearby. Under those conditions, statistically it was probable that most symptoms would be diving related, encompassing decompression sickness.

Nowadays, many of the diving population more closely resembles the general practice population. This includes a wide age range, with many people having a variety of illnesses, which can develop in relationship to, or coincidentally with, the diving activities. Thus it is essential to question every diving accident regarding their past medical history.

A similarly with medication usage. The diving population now has a much wider exposure to both therapeutic and other medications, which can produce side effects that can mimic decompression sickness. Typical medications include parasympathomimetic drugs, anti-malarials, carbonic anhydrase inhibitors and many others.

2 *A trial of pressure (recompression therapy) will clarify the diagnosis*. It often does not. Often the situation in which the recompression therapy is administered will result in an improvement in a variety of conditions. This case is an example. A more typical example would be the paraesthesia associated with anxiety and hyperventilation.[2] This affects up to 6% of the Australian population. With the increased density of air in the chamber, and often the restriction to breathing from masks, the hypocapnoea produced paraesthesia may be rectified in the chamber environment. A third example is hypoxia from any cause, including salt water aspiration.

Also, especially now that multiple treatments are given, the effect of time may ameliorate many acute illnesses. Most people get better from other illnesses, as well as from decompression sickness, with time.

3 *Exposure to altitude will aggravate decompression sickness*. Although this may certainly occur, especially if there are persisting tissue bubbles, it is unlikely to be a factor if adequate oxygenation has been given and the patient rendered free of bubbles and denitrogenated. Nevertheless, exposure to altitude can aggravate any neurological manifestation[2] for many reasons (hyperventilation, hypocapnoea, alkalosis, hypoxia).[3]

Conclusion

Despite the oft quoted diving medical dictums it may be more relevant, now that diving physicians have to cope with a general practice type population, to give more credence to traditional approaches to medical diagnosis.

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SPUMS NOTICES

SPUMS DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

New guidelines for candidates for the Diploma of Diving and Hyperbaric Medicine are being developed and will appear in the September 2000 issue of the Journal.

All those who have applied for permission to attempt the Diploma, and those who wish to apply, are asked to contact the Education Officer, Dr David Doolette at the Department of Anaesthesia and Intensive Care, The University of Adelaide, Adelaide, South Australia 5005, either in writing or by e-mail as soon as possible so that their names and particulars may be registered correctly.

Dr Doolette's telephone number is + 61-(0)8-8303-6382 and fax is + 61-(0)8-8303-3909. E-mail <David.Doolette@adelaide.edu.au>.

Current guidelines appear below.

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 evidence of satisfactory completion of examined courses in both Basic and Advanced Course in Diving and Hyperbaric Medicine at an approved institution.

3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months full time training in an approved Hyperbaric Medicine Unit.

4 The candidate must submit a written research proposal in a standard format for approval by the Education Officer before commencing their research project.

5 The candidate must produce, to the satisfaction of the Education Officer, a written report on the approved research project, in the form of a scientific paper suitable for publication.

Additional information

The candidate must contact the Education Officer to advise of their intended candidacy, seek approval of their courses in Diving and Hyperbaric Medicine and training time in the intended Hyperbaric Medicine Unit, discuss the proposed subject matter of their research proposed, and obtain instructions before submitting any written material or commencing a research project.

All research reports must clearly test a hypothesis. Preference will be given to reports of original basic or clinical research. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered.

It is expected that all research will be conducted in accordance with the "Joint NH&MRC/AVCC statement and guidelines on research practice" (available at http://www.health.gov.au/nhmrc/research/nhmrcavc.htm). All research involving humans or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. It is expected that research project and the written report will be primarily the work of the candidate.

The Education Officer reserves the right to modify any of these requirements from time to time.

Key words
Qualifications.

MINUTES OF THE SPUMS EXECUTIVE COMMITTEE TELECONFERENCE
held on 2000/2/13

Opened at 0800 Eastern Standard Time

Present

Drs R Walker (President), G Williams (Immediate Past-President), C Meehan (Secretary), P Dupont (Treasurer), J Knight (Editor), D Griffiths (Education Officer), C Acott (left 0915), S Mitchell, D Walker (left 0845), (Committee Members), M Bennett (ANZHMG Representative), V Haller (Co-convener 2000 ASM).

In attendance

Mr Steve Goble (SPUMS administrator).

1 Minutes of the previous meeting (1999/12/9)

Moved that the minutes be accepted as a true record. Proposed, John Knight, seconded, Peter Dupont, carried.

2 Matters arising from the minutes

2.1 Job description of the Convener was discussed and minor changes suggested.

2.2 Update on the SPUMS website.
The administrator is coordinating updating the website with the programer. There seems to still some problems getting the Diving Doctors List (DDL) on the website.

2.3 SPUMS dive medical on website.
This is planned as soon as possible.

2.4 All risks insurance policy for SPUMS equipment.
All committee members need to send full details of electronic equipment to secretary in order to insure them.

2.5 Education Officer/Board of Censors.
Dr D Griffiths' resignation has been received. David Doolette will be the acting education officer until the next committee meeting at the Annual General Meeting (AGM) in May.

2.6 Job conditions of the administrator.
This is being attended to by Dr R Walker and has been circulated. The committee has discussed and minor changes suggested.

2.7 Update on Indexing the Journal.
The Editor will explore other indexing agencies.

2.8 GST update. There is further information to come in.

2.9 The AGM minutes were discussed and will be posted at the AGM.

2.10 SPUMS involvement with an UHMS meeting proposed in Sydney.
This is to be discussed at the next face to face meeting.

3 Annual Scientific Meetings

3.1 1999 ASM, Layang Layang.
Final figures for profit and loss are still to be provided.

3.2 2000 ASM, Castaway Island, Fiji.
The program is complete. Detailed financial report should be available at the next meeting. Peter Dupont to request a written statement from Allways detailing financing of the ASMs.

3.3 2001 ASM, Madang, PNG.
The meeting will probably be the last week of May 2001. We will endeavor to avoid an overnight stay in Port Moresby. Topics discussed were "diving and the lung", "drowning and near-drowning". It was decided to have 2 overseas speakers to encourage lively debate.

3.4 Future ASMs.
This will be discussed further in Fiji.

4 Treasurer's Report

The Treasurer needs to set the subscription fee for next year. The administrator should provide the Treasurer with his financial accounts before each meeting so it can be circulated to the Committee.

5 Correspondence

5.1 Letter Bob Thomas re Industry Code of Practice, Division of Workplace Health and Safety. Dr R Walker will write to DWHS restating our position.

5.2 Letter Dr Deon Viljoen requesting recognition of overseas training to be on DDL. This has been passed onto Dr David Doolette.

5.3 E-mail Dr Knight re ASM costs. Dr Dupont will request a written statement from Allways detailing the ASM costs.

6 Other Business

Closed 0930

SPUMS

ANNUAL SCIENTIFIC MEETING 2001

will be held from
May 26th to June 2nd 2001
in
Madang, Papua New Guinea

Guest speakers
Dr James Francis and Dr Craig Conoscenti

CONSTITUTIONAL CHANGES

The Annual General Meeting at Castaway Island on May 13th 2000 passed the motions detailed below to amend the Statement of Purposes and Rules of the Society.

That the heading Board of Censors on page 19 of the Statemment of Purposes and Rules be changed to *Academic Board*.

That Rule 42 be changed by replacing the existing wording with *The Committee will appoint an Academic Board headed by the Education Officer*.

That Rule 42 (a) be changed by replacing the existing wording with *The make up of this Board will comprise individuals with proven clinical, scientific and research skills in the fields of diving and hyperbaric medicine. The minimum number of Board Members will be the Education Officer and two others.*

That Rules 42 (b) and 42 (c) be amended by removing the words *of Censors* from both rules.

The amendments will not come into effect until approved by the general body of members.

Any member who objects to the amendments should notify the Secretary of SPUMS, Dr Cathy Meehan, C/o Australian and New Zealand College of Anaesthetists, 630 St Kilda Road, Melbourne, Victoria 3004, Australia, in writing, before September 1st 2000. If any member objects a postal ballot will be held. If no objection is received it will be assumed that the membership has voted in favour of the amendments.

Cathy Meehan
Secretary of SPUMS

Key Words
Constitutional amendments

OZTeK2000

Saturday July 8th and Sunday July 9th 2000

The Australian Diving Technologies Conference is on again!
In association with the Boat Show and Dive Victoria Expo.
At the Melbourne Exhibition Centre
The premier Diving Conference of the region for 2000.

Start at the Polly Woodside Melbourne Maritime Museum with Happy Hour ($25.30).
Forum Sessions ($44 each), 0900-1230 and 1330-1700. Lecture night on Saturday ($22).
Silver Day Pass (One day 0900-1700 with admission to Exhibitions) for $ 77
Attend the Conference Dinner at the Centra Hotel on Sunday night ($49.50 food only).
Gold Conference Pass includes Full Conference and Workshops, Evening Lecture, Rebreather Try Dive and Exhibitions entry ($154)
Platinum OZTek 2000 Pass, all the benefits of a Gold Pass with Friday night drinks and the Conference Dinner ($220).

Forums will be on
Wreck Diving & Cave Diving Explorations
Equipment Developments & Applications
Decompression Theory & Decompression Illness
Rebreather Technology & Try Dives, Dräger (Ray & Dolphin), Inspiration, SM1600, PRISM.

There is no enough space to list the invited speakers, from Australia and Overseas. You will have to come and see and hear for yourself !

For further information, tickets and accommodation packages contact
OZTek2000, PO Box 894, Willoughby, NSW 2068.
Phone 0500 834 269. E-mail <tdi_aust@compuserve.com>

SPUMS

ANNUAL SCIENTIFIC MEETING 2001

will be held from

May 26th to June 2nd 2001

in

Madang, Papua New Guinea

Guest speakers
Dr James Francis and Dr Craig Conoscenti
Convenor Dr Guy Williams
Theme
Diving and the Lung
Workshop
Drowning/Near Drowning

Members wishing to present papers should contact
Dr Guy Williams
PO Box 190 Red Hill South
Victoria 3937, Australia
Tel + 61-(0)3-5981-1555 Fax + 61-(0)3-5981-2213
E-mail <guyw@surf.net.au>

Official 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>

HTNA Y2K

8th Annual Scientific Meeting on
Diving and Hyperbaric Medicine

Mercure Inn, Brisbane, Australia
6th-9th September 2000

Presented by the Hyperbaric Technicians and Nurses Association with the Australian and New Zealand Hyperbaric Medicine Group

Guest Speakers will include
Professor William Zamboni, Las Vegas, Nevada
Dr Simon Mitchell, Brisbane, Australia
Valerie Larson-Lohr, San Antonio, Texas
Richard Durnford, Seattle, Washington

For further information contact HTNA
Phone +61-(0)7-3371-6033
Fax +61-(0)7-3371-1566
E-mail <htna.y2k@wesley.com.au>

EUBS 2000

26th Annual Meeting of the EUROPEAN UNDERWATER AND BAROMEDICAL SOCIETY on Diving and Hyperbaric Medicine

to be held at the
Westin Dragonara Resort Hotel Malta

Thursday 14th to Sunday 17th September 2000
(Official language English)

Main topics

Pressure physiology and medicine
Management and treatment of diving accidents
Diving Safety
Marine Medicine
High pressure physiology
The future of Baromedicine in Europe
Hyperbaric Oxygen therapy
Gas physiology
Physician training
Cost-benefit in HBO_2
New indications for HBO_2
Principles of diver fitness.

Congress Secretariat
Alpine Travel Ltd.,
Alpine House, Naxxar Road,
San Gwann, SGN 08, Malta.
Tel + 356-387-361 (extension 208).
Fax + 356-389-627.
E-mail <eubs2000@alpine.com.mt>

PRE-CONFERENCE SEMINAR

Monday 11th to Thursday 14th September 2000
at the
Medical School of the University of Malta

Introductory Course in Diving Medicine

Course Director Professor David Elliott

For further details contact
Dr R Cali-Corleo,
Hyperbaric Unit, St Luke's Hospital,
G'Mangia,
Malta.

Tel + 356-371-849. Fax + 356-383-061
E-mail <irocali@daneurope.org>

LETTERS TO THE EDITOR

SPECIAL INTEREST GROUP
IN
DIVING AND HYPERBARIC MEDICINE

Australian and New Zealand College of Anaesthetists
Ulimaroa, 630 St Kilda Road
Melbourne, Victoria 3004

2000/5/29

Dear Editor

Membership of SIG in Diving and Hyperbaric Medicine

Due to various administrative problems, it appears that some people who have expressed an interest in joining the SIG have not received a response to their enquiry.

I write to ask that you place a notice in SPUMS Journal to request all those SPUMS members, whether or not they are anaesthetists, who wish to join the SIG, and have not heard from the College, to please write again to

Ms Helen Morris at the
Australian and New Zealand College of Anaesthetists
630 St Kilda Road, Melbourne 3004.
Tel +61-(0)3-9510-6299
Fax +61-(0)3-9510-6786
Her e-mail address is <helen@anzca.edu.au>.

I apologise for this administrative oversight. I look forward to welcoming you on board and to receiving your contributions.

Robert M Wong
Chairman
Diving and Hyperbaric Medicine SIG

Key Words
Meetings, qualifications, training.

BOOK REVIEWS

NEAR DROWNING: 47TH WORKSHOP OF THE UNDERSEA AND HYPERBARIC MEDICAL SOCIETY.
C W Dueker and S D Brown, Chairmen.
Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, Maryland 20895, USA. Published 1999.
Price $US 20.00. Postage and packing extra. Credit card orders may be placed by phone on +1-301-942-2980 or faxed to +1-301-942-7804. E-mail <uhms@uhms.org>.

Held before a small but erudite audience this one day workshop report maintains the high standards that we have come to expect from these UHMS publications. The morning session was devoted to the pathophysiological mechanisms of near drowning and then the management of the victim was covered during the afternoon.

In the introduction Dueker points out that drowning ranks as the 20th most common cause of death worldwide and although this is well behind heart disease and traffic casualties it is still well ahead of war injuries. He also notes that not everybody who dies in the water drowns but, certainly with scuba diving, drowning may well provide the final common pathway. Dueker describes well the pyramid of management of near drowning with education forming the base and intensive care the apex and then the factors that influence and help to prevent near drowning.

Carl Edmonds, in his usual inimitable style, presents a paper describing the pathophysiological mechanisms of drowning. In it he evaluates the research that has been undertaken and points out a number of misleading assumptions that have flowered over the years. The point he does make is that death by drowning is due to a progressive or irreversible pulmonary damage caused by progressive surfactant damage despite rescue, pneumonitis from the aspirated fluid or from vomitus, infection, and pulmonary oxygen toxicity from the attempted resuscitation. He concludes this chapter with a good review of the salt water aspiration syndrome, a classical case of which I saw only the other day.

Dr Claes Lundgren then discusses his research on the protective effect of the diving reflex in near drowning and concludes that "the diving response is likely to play a crucial role in the in the survival of drowning incidents, although current information does not allow us to account fully for how nature deals with the threat of hypoxic damage after the diving response is established but before metabolism is sufficiently suppressed by hypothermia."

Dr Chris Dueker then annotates a number of the common myths in near drowning such as laryngeal spasm resulting in death without water ever entering the trachea, the role of the Heimlich manoeuvre and the protective effect of very cold water.

Most of what Dr Carl Edmonds said in his next presentation on drowning with SCUBA has been previously reported in the SPUMS Journal and the MJA but it is satisfying to have it all in one compact form that can be easily referenced.

Mr Dennis Graver opened the batting after lunch with a discussion of the open water rescues and field resuscitation of the near drowned diver. He gives some examples of the changing philosophy in resuscitation over the years and opines that ventilation via a pocket mask remains today's preferred method of aquatic rescue breathing. This is an excellent paper that should not be hidden away but should be widely distributed amongst and read by the diving population. There is no excuse for any diver who does not know the skills needed to rescue and provide correct first aid to another injured companion. Any diver without these skills and knowledge should not be in the water.

A former SPUMS guest speaker at the Maldives, Bill Hamilton then spoke of the problems of near drowning in the technical diving field with the complicating factors of a possibly massive decompression obligation or oxygen toxicity.

Hospital management of the near drowning victim was discussed by Brown and Piantadosi who covered both the general and intensive care aspects of management.

Before the final discussion Chris Dueker spoke on prognosis for the near drowning victim. He noted that there are three valuable prognostic physical findings. Loss of consciousness signifies serious immersion but the converse does not necessarily apply. Pearn's "time to first gasp" is probably still the most reliable prognostic sign, and the presence of a pulse is a sign that recovery should be possible. The aim of rescue and resuscitation is to interrupt the hypoxic spiral before cardiac arrest occurs.

I am disappointed that I was not able to be present at this workshop as the report winnows an enormous crop of data eliminating much of the chaff. This report should sit on every medical library bookshelf and be read by every medical person who goes within cooee of the water be it pool, ocean or river.

David E Davies

Key Words
Book review, drowning, physiology, rescue, resuscitation, treatment.

HIGH PRESSURE BREATHING AIR HANDBOOK

William E McBride.
ISBN 0-655986-0-8

Review copy from Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Price $US 39.95. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

The title suggests that this book should be a source of information on all aspects of the field. I do not think it fills this role as it lacks detail on some topics and was of no help in solving the problems I hoped it would answer. The author was/is a compressor system designer and installer and the book is good on the areas that he is likely to be interested and skilled in. So, if you are about to buy a new compressor for your hospital or dive shop you probably should buy it. If you run a chamber or shop there may be enough in it for you to consider buying it. If you are a typical SPUMS member, with interests in diving medicine, fitness for diving and that sort of thing, there is probably not enough in it to make it worth your money.

It is good on compressor specification and using the tendering process to get what you want in an installation, and how to avoid mistakes by not being clear in the specification. It is also useful on choosing the best size of compressor and storage bank. The safety of gas storage, filling stations and of cylinders is well covered. Because it is written for the USA market it is less relevant to other countries on the legal requirements. Also, because of the relative shares of the market, it gives more attention to the needs of fire departments. They use compressed air for fire fighting and rescues. Diving gets less consideration and newer topics, like the production, compression and storage of Nitrox mixtures get no mention.

Unfortunately it was of little help on high-pressure air related problems. We recently discovered a valve seat in a high-pressure airline had burnt out. Some engineers said heating caused by rapid air compression was the likely cause. Others said no; the valve must have been contaminated for the seat to burn. I went to the book and found no information on valve heating or contamination of gas lines. I also looked for information on the methods of cleaning the piping to remove any contamination or residue of combustion products. I could find no information on this either. Information in the book is not easy to find because there is no index.

John Pennefather
Scientific Officer
Submarine and Underwater Medicine Unit
Royal Australian Navy

Key Words
Air, book review, equipment.

STARS BENEATH THE SEA
The extraordinary lives of the pioneers of diving.

Trevor Norton.
ISBN 0 7126 8072 1. October 1999. Hardback.
Century, 20 Vauxhall Bridge Road, London SW1V 2SA, UK
RRP in the UK £12.99, in Australia $35.00

Anybody who has ever dipped a toe in the water has heard of Hans Hass and his adventures in the Red Sea. Fewer might have heard or read about William Beebe or Frederic Dumas. All diving medicos have heard of John Haldane and possibly his son. It is only the more serious students of diving history who have heard of Guy Gilpatric, Louis Boutan, Ernest Williamson and Peter Throckmorton. Each of these characters, and more, rate a chapter in a recently published book from the pen of Trevor Norton.

The book is written as if the author has personally known each of his subjects. It is filled with anecdotes about their personal lives, their ingenuity and courage to enter territory, which to that time was quite unexplored. How can you fail to warm to people who drink a pint of acid to change the pH of their blood, deliberately ascend from 100 m without benefit of breathing apparatus or patent a giant rubber octopus in case anyone else feels the need for one? They were all brave, brilliant and quite barmy.

Trevor Norton is an enthusiastic narrator whose book is filled with humour and much fine detail. He is the Professor of Marine Biology at the University of Liverpool and Director of the Port Erin Marine Laboratory on the Isle of Man. In the mid 1950s he first went underwater at the age of about 15. As he is a marine ecologist much of his research involves diving.

He first became interested in the history of diving by accident when, for fun he gave some students a lecture on the subject and was asked to give a version of it to inaugurate a conference for the Society for Underwater Technology. This went down so well that he was asked to tour Britain with the lecture. He realised then that he was being considered an expert in a subject about which he claimed to know next to nothing. So, out of embarrassment, he began to research the original books and papers and simply fell in love with the characters.

Stars Beneath the Sea has made the bestseller charts in Britain and was very kindly reviewed both by the national papers and the specialist press in the UK. The paperback edition comes out in May 2000, as already there have been two reprintings since the original publication in October 1999.

The author has now just completed another book that relates the whole story of the dives and misadventures of Jack Kitching, set against the wonderful scenery, history and natural history of South West Ireland. Again it is an

unbelievable and amusing tale of extraordinary people doing extraordinary things.

Stars Beneath the Sea is very easy to read and has the potential to become a best seller in Australia just as it did in the UK. I heartily recommend it for all libraries, both personal and public.

David Davies

Key Words
Book review, diving operations, history, general interest.

THE HISTORY OF AMERICAN DEEP SUBMERSIBLE OPERATIONS

Will Forman
ISBN 0-941332-72-1
Best Publishing Company, P.O.Box 30100, Flagstaff, Arizona 86003-0100, U.S.A.
Published 1999. Price from the publishers $US 39.50. Postage and packing extra. Credit card orders may be placed by phone on +1-520-527-1055 or faxed to +1-520-526-0370. E-mail <divebooks@bestpub.com>.

While Man has been to the moon many times both in person and by using robotic machines, man has only visited the deepest ocean on one occasion, which was the *Trieste* dive in 1960. Currently, no United States manned submersible can dive deeper than the *Alvin*, rated to about 3,900 m (~13,000 feet). *Seacliff* had the capability to dive to 20,000 feet (~6,060 m), but has recently been decommissioned and sent to Woods Hole. It is rumoured that the pressure hull from *Seacliff* will be fitted to *Alvin* to extend human reach, once again, to about 20,000 feet (~6,060 m). The sad record of the commitment of any government to manned exploration of the deep ocean is well documented in this great book.

This book is a long awaited treasure trove of words and pictures for anyone interested in the deep ocean. The author, Will Forman, offers the insight that only can be presented by one intimately involved with the small and exclusive world of deep ocean submersibles. Will Forman worked on many of the projects and had the contacts to know all there is to know about the era of American Deep Submersibles from 1959 until 1995.

The history contained in the first chapter is thorough and well researched and has many interesting details from the 1775-1930's period. Lieutenant Payne of the American Confederate Navy in about 1860 seems to have set some sort of record for surviving submarine disasters in the submersible vessel *Hunley*. After two escapes Payne seems to have looked elsewhere for duty. The *Hunley* provided the first free ascent from an American submarine, when Lieutenant Hasker, waited until the pressure equalised before opening a hatch at 42 feet (12.7m). Such interesting detail and stories make this book enjoyable to read, rather than just a catalogue of engineering facts and log of dives.

The initial chapter includes many early submarines, starting in 1775 with the *Turtle*, to provide an historical perspective, but by the end of the first chapter with William Beebe in 1954, the submarines end and the succeeding chapters focus on the true deep submersibles, vessels of adventure and research.

It was a bit inconvenient at times to not have other nations' submersibles listed, but I should not have expected this from the title of the book.

Last October I was lucky enough to be given the opportunity to dive in one of the submersibles included in the book. I went to sea off San Diego with the USN and the DSRV *Avalon*. We planned to dive to Deep Seat, a mock submarine hatch located at 1,950 feet (591 m). Unfortunately topside called off the dive during our final approach, because of deteriorating weather, so we did not, as planned, lock onto Deep Seat, de-water and stand on the bottom of the sea. I have looked out of many dive bells and even a Pisces Submersible in the North Sea. But as you drop below 1,000 feet (~300 m) diving towards 2,000 feet (~600m), and enter the deep ocean, it was a thrill of a lifetime to me to be allowed to travel to a depth that few have the chance to visit (1,934 feet or ~586 m the certificate says).

While I admit to being an unashamed enthusiast of the deep ocean, this book is a must for all who regard the deep ocean as the next, or even the forgotten frontier. It explains so much about the why, and the why not, that has restricted man in his attempts to explore our own planet. If only some American President had supported a wet version of NASA, but that is not as sexy as the moon.

American deep Submersible Operation 1775-1995 is not only a great documentation of engineering feats and facts, it is also a great read for the many embellishing and at times amusing stories that make the book come to life. 312 big pages with everything you could wish to know on the subject.

Bob Ramsay

Key Words
Book review, deep diving, equipment, general interest, history, submarine.

This book is available to members of the Diving Historical Society Australia, South East Asia though DHS ASEA.

SPUMS ANNUAL SCIENTIFIC MEETING 1999

WHAT IS THE OPTIMAL TREATMENT "DOSE" FOR DECOMPRESSION ILLNESS?

Alf Brubakk

Key Words

Decompression illness, oxygen, treatment.

Background

The basis of any treatment of decompression illness (DCI) is an increase in pressure and oxygen content of the breathing gas. However, the optimal way to combine these has not been determined. Results of treatment vary considerably from centre to centre and there are few studies that actually compare the effect of different treatment algorithms in patients with similar symptomatology.

Even if many details regarding the pathophysiology of decompression sickness still are unknown, there is little doubt that the basic problem is the formation of bubbles in the living body. The formation of bubbles requires supersaturation, that the gas tensions in the tissue are above that of the environmental pressure. Treatment has, till now, been focused on this and is intended to reduce the volume of the gas phase and eliminate the excess inert gas load. There is, however, no practical way to measure this load. So the effect of treatment has been judged using clinical signs and symptoms as an end point. This has, however, diverted our attention from the fact that reducing the volume of the gas phase and eliminating inert gas are two different processes that have quite different time constants.

When pressure is applied, the volume of the bubble is immediately reduced according to Boyle's law. Thus, if the bubble itself has a mechanical effect on a tissue, increased pressure will immediately relieve the symptoms. The elimination of gas, however, is a much slower process, in particular if bubbles are present.[1] The bubbles trap gas and reduce the tissue gas tension, which increases the elimination time for the gas.[2] Furthermore, as gas has to be transported to the lungs by the blood, circulatory factors can play a significant role in determining the elimination time. Supersaturation and gas bubbles can be present for hours in the tissue, leading to secondary effects of bubbles that may eventually influence the final clinical outcome.

Different treatment procedures

When oxygen and pressure are used for treatment, there will be the following effects:

1 Increase in environmental pressure. This will reduce the size of the gas bubble and reduce the risk of ischaemic damage.

2 Increase in oxygen partial pressure in blood and tissue. This will increase the gradient for inert gas removal.

3 Increase in the oxygen content of arterial blood. This will increase the oxygenation of the tissue, thus reducing the risk of hypoxic damage.

4 Biochemical and reactive effects of oxygen. These effects, although the least understood, may be highly significant in the treatment of DCI.

Many different treatment regimens have been tried over the years. These procedures are usually based on clinical experience and few studies have been performed actually testing their effectiveness in different groups of patients.

Recommended treatment pressures vary from 200 to 780 kPa (2 to 7.8 bar or 10 to 68 m), while oxygen tensions vary from 180 to 300 kPa (kPa and bar are used in this paper as absolute, not gauge, measurements). However, as was pointed out in a recent workshop,[3] compression to 18 msw (280 kPa or 2.8 bar) breathing 100% oxygen is the only procedure where extensive clinical experience exists. Therefore this should probably be the basic treatment in all cases. In practice, this means the use of USN Table 6 (USN6). However, several studies have documented that both shorter tables at the same depth[4] and treatments at 200 kPa (2 bar or 10 m)[5] give equally good results. Recently, this last group published that 70% of the divers with neurological symptoms were symptom free after two to six hours at 200 kPa (2 bar or 10 m) and that 13% of these divers had persistent manifestations after one month.[6] Another point that is worth noting is that USN6 is used differently in different centres. The number of oxygen cycles vary, some centres use a short, deep pressure spike before commencing with this table and some centres use oxygen on the surface following the end of treatment.

There is little data to support higher treatment pressures. However, most people with experience in the field have case histories where a patient who showed no improvement at 280 kPa (2.8 bar or 18 m) improved on reaching 600 kPa (6 bar or 50 m), either breathing air or a nitrogen/oxygen mix.

Treatment at 600 kPa (6 bar or 50 m) used to be the recommended treatment for air embolism. The theoretical basis for this is that an increase in pressure will reduce bubble size. However, the reduction in bubble size is greatest at the first doubling of pressure (100-200 kPa, 1-2 bar or surface to 10 m). Indeed, Gorman et al. showed, in rabbits, that the vascular bubbles in the brain were cleared as effectively using 202 kPa (2.02 bar or 10.2 m) as using

pressures up to 1,010 kPa (10 bar or 90 m).[7] Kunkle and Beckman showed that bubble resolution time would decrease by a factor of two if oxygen at 280 kPa (2.8 bar or 18 m) was used instead of oxygen at the surface and that further increase in pressure would not decrease resolution time further.[8] Monitored bubbles in the pulmonary artery we were also able to show this and further noted that there was no difference in bubble elimination time in the pressure range from 200-400 kPa (2-4 bar or 10-30 m).[9] In performing these studies, we were impressed by the effectiveness of recompression to 200 kPa (2 bar or 10 m) even using air. Animals with a large numbers of gas bubbles, with hardly any heart beat and no respiration, recovered immediately on arrival at pressure, demonstrating clearly, that in these cases, compression of the bubbles was lifesaving. Furthermore, histological studies of these animals central nervous systems showed that only one out of seven animals had any damage (Brubakk et al. unpublished).

One important point that is often disregarded in evaluating treatment procedures is the ability of the procedure itself to produce supersaturation and gas bubbles. Oxygen in excess of metabolic needs will increase total gas tension and procedures using inert gas mixes can add to the inert gas load. Thus it is conceivable that a procedure that initially removes the gas bubbles, actually may produce new gas bubbles during ascent to the surface.

During recent years, there has been considerable discussion about the use of helium/oxygen mixtures, mostly the use of 50/50 heliox at 400 kPa (4 bar or 30 m) (COMEX 30).[10] This procedure was developed by a French diving company (Comex) who claim to have excellent results with this approach. There are several differences between this approach and USN6, namely a higher environmental pressure (400 vs 280 kPa), a reduced oxygen tension (200 vs 280 kPa), longer oxygen breathing times at greater depths and the use of helium. It is very difficult to judge which of these factors play a significant role for the treatment outcome.

The dose of oxygen has only been considered to a limited degree when evaluating treatment procedures. In general, there is a belief that more oxygen is better and that the only limitation is oxygen toxicity. Oxygen is a vasoconstrictor and, at oxygen tensions of about 200-280 kPa (2-2.8 bar or 10-18 m), blood flow to all organs will be reduced by approximately 20-25%.[11] Furthermore, as oxygen tensions increase, the shunt fraction through the lung will increase, thus reducing the effect of higher oxygen tensions.[12] The use of lower oxygen tensions may actually also be of benefit. Leitch and Hallenbeck, in 1985, showed that oxygen at 200 kPa (2 bar or 10 m) was the optimal treatment gas in spinal cord decompression sickness.[13]

More importantly, oxygen at pressure has numerous biochemical effects which may be of importance when judging the optimal dose of oxygen. If indeed vascular obstruction and endothelial damage plays an important role in decompression illness, decompression illness may be compared to reperfusion injury. Blocking leucocyte adhesion[14] and C5a activation[15] by monoclonal antibodies significantly reduce the injury after ischaemia and reperfusion. In these situations reactive oxygen species[16] play a significant role and it is reasonable to assume that the correct dose of oxygen is important for successful treatment. For example, it has been demonstrated that the glucose metabolism in the injured brain improve after 35-40 minutes at 150 kPa (1.5 bar or 5 m) oxygen, but deteriorated after 15 minutes exposed to 200 kPa (2 bar or 10 m).[17] Timing of treatment as well as the tissue at risk probably also plays a role.

Thom et al. have shown that a single 45 minute exposure to an oxygen tensions of 280 kPa completely blocks activation of leucocytes, a mechanism of central importance in tissue injury and endothelial damage,[18] and this effect lasts for up to 8-10 hours.

End point of treatment

In most situations, the end point of treatment is the elimination of clinical symptoms. Sometimes this can be pretty obvious, as in a patient with a severe paralysis who is able to move his legs. In other cases, it may be much less clear and the treatment results may be influenced by the skill and thoroughness of the examining doctor. It is also well documented that even severe damage to the spinal cord can leave few symptoms.[19]

What is "treatment dose" ?

This is not an easy question to answer. As is pointed out above, there are numerous treatment variations, which make it difficult to compare different treatment algorithms. One simple way of defining this would be to simply integrate the treatment profile. For a single treatment this could be

ppO2 * Environmental Pressure * time (Bar^2 * min).

Using this formula a single USN6 treatment would have a dose of approximately 1,133, while a Comex 30 will give a dose of 2,187, or about 90% higher.

This calculation is an oversimplification and does not, in any way, take into consideration differences in the effects of pressure and oxygen, the possible importance of using different inert gases or the effect of air breathing intervals or ascent rates. It does, however, illustrate nicely the considerable differences between two treatment procedures. For example, breathing oxygen at surface for 120 minutes, will give a dose of 120. This treatment, when initiated at the time of maximum bubble formation, removed

all gas bubbles and was able to prevent serious decompression sickness and CNS changes in a study in pigs (Brubakk et al. in preparation).

Where do we go from here ?

As is pointed out above, USN6 is considered the standard treatment for all cases of decompression sickness. It is however worth asking if this is a correct approach. The three most important questions that can be raised here are probably.

1 Will time to treatment influence the treatment procedure ?
2 Will the type of dive that initiated the symptoms influence the treatment used ?
3 Will symptomatology influence the choice of treatment ?

Recently, studies have claimed that time to treatment is not of importance.[20] The authors argue, based on their results, that patients should be transported to a large facility with sufficient medical resources. However all the patients in these studies had several hours between exposure and treatment.

There is sufficient data to support the assumption that very rapid treatment can reduce the effect of even serious decompression accidents. Surface decompression using oxygen is a well established diving technique, that apparently has few decompression problems.[21] In this procedure, the diver returns rapidly to the surface and is recompressed within 5 minutes to 12 msw (220 kPa or 2.2 bar). We have demonstrated in animals that this procedure produces a significant number of gas bubbles in the surface interval, which disappear during treatment. In many cases however, the bubbles reappear after treatment is ended, indicating that the treatment is sufficient for removing the initial bubbles, but that it is not adequate for eliminating all excess gas.[22]

A large number of successful in-water decompression treatments have been performed, using air, by going to 9 m deeper than the dive. This is remarkable and is perhaps due to the fact that treatment is performed shortly after symptoms appear.[23] We were able to demonstrate experimentally that short (70 minute) treatment at 200 kPa (2 bar or 10 m) using air was effective in animals, if treatment is initiated at the time of maximum bubble formation. The same study demonstrated that 100% oxygen at surface was equally effective under these circumstances. (Brubakk et al. in preparation).

Nearly all decompression tables have been tested by using decompression sickness as an endpoint. When decompression sickness occurs, the diver is immediately treated and it is generally assumed that he thus can escape serious injury. This argument has been used repeatedly by researchers seeking approval for their experiment from the ethical committees.

Rapid recompression will be effective in reducing the size of the gas bubbles and thus limit their direct, mechanical effects. However, considerably more time is needed for eliminating all excess gas. Some treatment procedures recompress the diver considerably deeper than the standard 18 msw (280 kPa or 2.8 bar). If reduction of bubble size is the aim, this does not make much sense as the relative size reduction of additional pressure increase is small.[7] We have shown that the elimination time for bubbles in the pulmonary artery is similar in the pressure range of 200–400 kPa (2-4 bar or 10-30 m).[9]

In a deep dive, particularly if helium is used, a considerable gas load will accumulate. To eliminate this gas load will require time. Thus it is quite conceivable that the length of the treatment, but possibly not treatment depth, may be dependent upon the primary dive.

Due to the difference in the speed of uptake and elimination of gas in the different tissues, it is likely that the gas load and thus the degree of bubble formation will be different in different tissues. A short, deep dive will produce bubbles in quite different tissues than will a long, shallow dive. This is in accordance with what was pointed out by Lanphier and Lehner,[24] that different dives produce different symptomatology. Central nervous symptoms are more common in deep, short dives, while long, shallow dives give predominantly symptoms from joints and muscle. Computer simulations support this and also indicate that bubbles from such deep dives disappear more quickly using pressures of 400 kPa (4 bar or 30 m) with 50% oxygen than when using USN6.[25]

The approach used by Comex for many years, where they treat minor symptoms at 220 kPa (2.2 bar or 12 m) and go to 400 kPa (4 bar or 30 m) for more serious symptoms, may actually have considerable merit.

I think, however, that the time to treatment is important and may influence the choice of table.

Conclusion

At present we do not have sufficient information to make adequate decisions about the optimal treatment "doses" of pressure and oxygen, but such information is urgently needed. This is especially important as we can expect new treatment challenges as divers, using rebreathers, are now able to go deeper, stay longer and use a number of gas mixtures.

Both clinical and experimental data indicate that even compression to 200 kPa (2 bar or 10 m) using air can be

effective if treatment is started early. This has to be further explored as it will have significant impact on the acute management of decompression accidents.

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WHY EXERCISE IS NOT ALWAYS GOOD FOR YOU TWO CASE REPORTS

Robyn Walker

Key Words

Carbon dioxide, medical conditions and problems, physiology, risk, unconsciousness.

Introduction

Royal Australian Navy Clearance Divers are required to pass stringent physical fitness standards, regardless of age. As a consequence, individuals often push themselves to their physical limits attempting to meet these standards, particularly during the selection tests to join this select and elite branch. At times individuals exhibit the mental toughness necessary to push through their own pain barriers, with devastating physical consequences. Two such cases are presented.

Case 1

Diver A was a 31 year old male, considerably older than most trainee divers, highly motivated, with a somewhat obsessional desire to become a Clearance Diver. He was well educated, fluent in a number of languages and had worked as a recreational dive instructor before enlisting in the Navy. He claimed that, in the two years before joining the Navy, he had performed approximately 1,600 dives without mishap. He was extremely proud of his underwater ability and was in good health, but said that he had never been a good runner.

He was a participant on the Clearance Diving Acceptance Test Course. This is a punishing course, where candidates are tested both physically and psychologically. The candidates do not undertake many dives during the course, which is designed more to assess teamwork skills and attitude during extreme situations. The course was being conducted in Pittwater, approximately 40 km north of HMAS PENGUIN, when the report came one Saturday night that he had collapsed. In fact this was the third time he had "collapsed", but this information had not been passed to senior medical staff.

The history I received that night was that he had collapsed during a PT session, which was said to be fairly testing. He was alleged to have lost consciousness, was frothing at the mouth, tachycardic, profusely sweating, with his eyes rolling back. It is at your peril that you ignore the statement of a senior diving instructor who says "Ma'am, he looked like he was going to die".

I was a bit alarmed by the story, however I was then informed some two hours had elapsed since his collapse and he had since made a good recovery. He had not dived for the 24 hours before his collapse, and in the previous week had only undertaken one 6-metre dive on air. He had complained to the training staff that whenever he had to run very hard, he became dizzy, his head spun, he developed blurred vision and difficulty breathing. His symptoms always resolved on completion of exercise.

By the time I saw him he had fully recovered and was normal to examination. An ECG showed sinus rhythm, with a few ventricular ectopics, which were considered to be benign. An echocardiograph revealed no structural abnormality. A maximal exercise stress test was performed; he exercised for 17 minutes, and only stopped because of calf pain. When he exercised, the number of ventricular ectopics decreased. A malignant arrhythmia was not considered to be a likely explanation for his symptoms.

Routine spirometry, lung volumes, transfer factor and arterial blood gas measurements were all normal. He then went on to a progressive cardio-respiratory exercise test. He exercised well, to 200 watts, but went into respiratory failure during the test. Despite a substantial fall in arterial oxygen saturation, to a PO_2 of 43 mmHg, and an increase in end-tidal PCO_2 to 63 mmHg his respiratory rate remained at 9 breaths per minute. By the end of the test his symptoms of blurred vision and dizziness were reproduced.

His ventilatory responsiveness to carbon dioxide was then tested. He had a reasonable response to carbon dioxide, but the pattern of his hyperventilation was unusual, with an increase in tidal volume but no accompanying increase in frequency. It appeared that he was voluntarily holding down his respiratory frequency. The patient then admitted that he was extremely proud of his ability to skip breath and said that for all his 1,600 dives he only took three breaths a minute. The respiratory physician said that he did not believe the patient's voluntary reduction in respiratory frequency presented a hazard to him diving, although he becomes significantly hypoxaemic and hypercapnic at the extremes of exertion. I disagree with this opinion for a number of reasons.

The normal response to hypercapnia is an increase in respiratory rate and the development of a headache. These symptoms can cause significant distress. It is known that carbon dioxide retainers have a higher threshold before experiencing the effects of raised carbon dioxide partial pressure. Our patient's threshold was much increased, so he did not experience those warning signs. It is also known that hypercapnia may potentiate oxygen toxicity and nitrogen narcosis, may precipitate oxygen convulsions and, by itself, has been associated with loss of consciousness.[1,2]

Situations likely to aggravate hypercapnia include exercise, an increased breathing resistance, use of carbon

dioxide absorbent systems, hyperoxia and nitrogen narcosis. These are conditions faced everyday by a clearance diver diving with closed circuit rebreathers.

We considered this patient to be at increased risk of developing both carbon dioxide and oxygen toxicity when using closed circuit breathing sets. The patient believed he could "unlearn" his ventilatory response, however, we were unwilling to accept this as, in the face of extreme provocation during the exercise test, he had been unable to increase his ventilation even in the presence of life threatening acidosis (a consequence of his severe hypoxia and hypercapnia).

He was made permanently unfit to dive using closed circuit diving sets, which meant he could not be a Clearance Diver in the Navy. His fitness for open circuit diving poses an interesting problem, particularly in view of his uneventful diving past. He was discharged from the Navy intending to resume his recreational diving instructor career.

Case 2

Diver B was a 24 year old male in good health, who had not dived before. He was a student on the Scuba Air Diving Course and had passed all the requirements of the Royal Australian Navy diving medical. He denied taking any medication or tablets on day 1 of the course. This was later determined to be inaccurate. He was in the last week of the three week course and was assessed as being one of the better students, performing well in both the diving and physical fitness activities.

B collapsed during a physical training session involving, while wearing overalls, a 200 m harbour swim followed by an 8 km run back to the Naval base. The history related by his course mates was that B had become distressed after running 3-4 km. He stopped, fell to the ground and then got up complaining of thirst. A fellow student took his pulse, which was said to be 180 per minute. Unfortunately, he was encouraged to continue by one of his classmates. He fell again. His classmate thought "We're holding everyone back" so he piggybacked the victim up the hill. At the top of the hill B was given a drink of water but did not feel much better. B continued the run, becoming increasingly distressed, but kept repeating, "I have to get home. I have to keep going."

When he reached the base, he suddenly sprinted but was veering from side to side on the road. He reached the Diving School, where his distress was immediately recognised and he was carried to the Submarine and Underwater Medicine Unit.

On arrival he was unconscious, with a Glasgow coma score of 7. He was in severe respiratory distress with a respiratory rate of 40. He had a sinus tachycardia of 180. He was peripherally shut down and sweating profusely. His temperature was 38.4°C on arrival and was never measured any higher. He was intubated, his ventilation controlled and intravenous fluids started. Arterial blood gases, while on 100% oxygen, revealed a profound metabolic acidosis (pH 7.17, PCO_2 43 mmHg, PO_2 371 mmHg, HCO_3 15.8 mmol/l, base excess (BE) –11.8 mmol/l and O_2 saturation 99.9%). He was transferred to the Royal North Shore Hospital.

A chest X-ray and CT scan of his brain were normal. His sinus tachycardia slowed with fluid replacement although he was also given adenosine. His clinical condition settled quickly and he was extubated later that afternoon.

He had, however, suffered a severe systemic insult as he went on to develop acute hepatic failure, disseminated intravascular coagulation and renal failure (alanine amino transferase (ALT) peaked at 12,000 U/l and aspartate amino transferase (AST) 9,830 U/l on day 3 post injury, platelets 79 x 10^9/l, the international normalised rate (INR) was 4.1 and creatinine peaked at 0.2 mmol/l). He developed widespread ST segment changes on his ECG. Creatine kinase peaked at 1,323 and was said to be of skeletal muscle origin. Despite this there was no evidence of rhabdomyolysis. Echocardiography and gall bladder ultrasound were reported as normal examinations. Serology for hepatitis A, B and C and HIV was negative. Urinary drug screens (including paracetamol) were negative although it was then discovered B had been taking creatine supplementation, in accordance with the manufacturer's guidelines (1 teaspoonful a day) in the two weeks before and during the course.

B eventually improved with conservative management, but not before the liver transplant team was consulted and were on stand by. At the time of discharge from hospital, some 14 days after the incident, all biochemical parameters had returned to normal except his ALT and AST. These had dropped considerably but it took another couple of months more before they returned to normal.

His discharge diagnosis was heat stroke, in association with severe physical exertion. Whether or not the creatine supplement played a part in his presentation was uncertain although the consultant physician believed B would be fit to return to complete his diving course within a few months.

There is some difficulty in accepting this diagnosis. Heat stroke, by definition is thermo-regulatory failure, usually associated with warm environmental conditions and core body temperatures over 41°C. While some sufferers can be sweating on presentation, more usually they present with dry skin, having ceased sweating. Contributing

factors include dehydration and antihistamines with anticholinergic activity. On the day of B's run the temperature was only 19°C and it was drizzling. His temperature was never recorded higher than 38.4°C. He had been well hydrated before the exercise that morning, he had no underlying illness and was on no medication, other than the creatine. He was accustomed to the level of exercise undertaken.

What about the role of creatine? Creatine is found naturally in skeletal muscle, the heart, brain, testes and other tissue. It exists in its free form (1/3), and as phosphocreatine (2/3). Creatine plays an important role in anaerobic ATP production during maximal anaerobic burst type activity. During intense muscle contraction, ADP is rapidly phosphorylated by phosphocreatine to produce ATP that is utilised within the myofibrils of skeletal muscle. This reaction can produce ATP for 10–20 seconds of maximal exercise, after which, other mechanisms are needed for ATP production (anaerobic glycolysis or aerobic oxidation of carbohydrates and fats).

The supposed basis of the ergogenic effect of creatine supplementation is an increased storage pool of phosphocreatine in skeletal muscle, an enhanced resynthesis of phosphocreatine during recovery periods after intense exercise and possibly an increase in skeletal muscle protein synthesis.

Typically creatine supplementation is a loading dose, of 20 g daily for 5 days, followed by a maintenance dose of 2 g a day. The loading regime can increase a person's total creatine stores by 17-22%. Without a loading phase a daily dose of 3 grams will achieve a similar increase after about 28 days.

Creatine is ergogenic, for repeated 6 to 30 second bouts of maximal, stationary cycling (recovery periods of 20 second to 5 minutes). It is not considered ergogenic for single or repeated swimming and running sprints, but it may increase strength as a result of increased protein synthesis in muscle. That is not yet proven. It does not benefit sub-maximal or endurance exercise. The individual response can vary greatly in the extent of increase in muscle creatine concentration, as well as the performance results.

There is limited data available regarding the safety of creatine.[3,4] It is not considered a drug, is distributed as a food agent and therefore claims regarding performance and safety do not need to be substantiated. It causes weight gain initially, thought to be as a result of water retention. There has been no reported relationship between creatine supplements and muscle dysfunction, or gastrointestinal symptoms. However, diarrhoea and gastrointestinal pain have occasionally been reported as side effects of the loading dose. Cramping, strains, tears and muscle tightness have been reported with creatine use. These effects have been thought to be associated with the water retention, which increases muscle compartment pressure leading to risk of muscle dysfunction. Increased levels of creatine have been measured in both serum and urine with supplementation, however, normal kidneys manage this load without compromise.

Short-term supplementation of 10 days or less has no effect on cardiac ejection fraction or blood pressure. Creatine is normally found in the brain and in the CSF. Some studies have suggested that there might be a relationship between seizure disorders and increased brain creatine. However it is not known whether oral creatine supplements affect brain levels. Creatine is also found normally in the testes where both forms are involved in sperm production. As endogenous creatine synthesis is suppressed during periods of supplementation it is postulated that Sertoli cell function may be affected. Long term effects of this suppression are unknown.

It is possible that in B's case the risk of dehydration was increased a consequence of creatine induced fluid shift into skeletal muscle.

B, although having made a complete physical recovery, was somewhat anxious about returning to rigorous exercise. He underwent psychological review and an individualised, supervised, PT program was arranged for him. Then he had a formal exercise test to prove to him, in the safety of a hospital environment, that exercise was safe. Resting lung function was normal. He achieved his maximum predicted exercise workload, at a maximum oxygen uptake (VO_2) of 96% of predicted and a heart rate of 90% of predicted. However, his maximum ventilation achieved was only 74% of predicted with a peak end-tidal CO_2 of 46 mmHg. His respiratory rate did not increase with exercise but he had an increase in tidal volume. This suggests reduced sensitivity to CO_2, as with Diver A, but milder. It was considered unethical to repeat exercise testing to the limits of exhaustion.

There remains the question of fitness to dive. Multi-system organ failure had followed routine exercise in a man with a mildly reduced sensitivity to carbon dioxide and who was taking creatine supplementation. In coming to a decision about his future we used similar criteria to Case 1. A person with reduced sensitivity to carbon dioxide is at increased risk of carbon dioxide toxicity, cerebral oxygen toxicity, nitrogen narcosis, and loss of consciousness. So B was declared to be fit for open circuit diving, but unfit for closed circuit diving. He has returned to exercise and to his ship at sea. But, at the time of writing, has not yet returned to repeat his diving course.

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ADJUNCTIVE THERAPY IN DECOMPRESSION ILLNESS: PRESENT AND FUTURE

Richard E Moon

Key Words
Decompression illness, drugs, treatment.

Historical background

When attempting to evaluate the value of adjunctive treatment, it is instructive to examine case descriptions from an era when none were available. In Edward Ellsberg's book, *Men Under the Sea*, is a dramatic description of air recompression therapy of a diver with a severe case of the bends, experienced during the salvage of the submarine *S-51* in 1925.[1]

> [After a 60 minute dive to 132 feet, and two hours of in-water decompression]…the tenders finished undressing the divers, leaving the *Falcon's* fantail a mess of wet lead shoes, lead belts, helmets, hoses, and sodden diving rigs, and the three, still in their underwear, hurried below for supper, already somewhat late.
>
> Five minutes later, seated on a bench before the heavily laden mess table, L'Heureux, still as merry as ever, suddenly collapsed, pitched forward on the table, unconscious!

No need to ask questions in that company. "The bends" and a bad case of it! No one wasted time in futile first aid measures. Hastily his shipmates seized the silent figure of L'Heureux, unceremoniously rushed him up the steep ladder to the deck above…

In through the round steel door of the recompression tank went L'Heureux's inert form, one diver dragging his head, another pushing on his legs. Running from the wardroom came Surgeon Flotte, to dive through the opening almost on L'Heureux's heels. The door clanged shut behind him. On went the compressed air, hissing gently into the chamber as was customary. Hastily Surgeon Flotte felt L'Heureux. No sign of heartbeat. The man was completely out, might die at any moment, might perhaps already be dead from bubbles of air clogging his heart. It was no time for routine measures. At any cost those bubbles must be reduced to a size small enough to pass through the heart valves, to allow circulation to continue. And only high air pressure could compress them enough for that.

Dr. Flotte sprang for the air valve and twisted it wide open (apparently, in those days the insider tender operated the chamber). Immediately the low hiss of the incoming air changed to a loud roar and, under the terrific pressure of the high pressure air banks, air started to pour into that recompression chamber. The needle on the caisson gauge jumped like a race horse getting away from the barrier, continued rapidly round the dial. Twenty pounds, 40, 50. Dr. Flotte's ears began to ring. That was as high in pressure as we had ever gone before on anybody. But no stop now. Sixty pounds. Blood oozed in Flotte's nose and mouth, but still he kept the roaring in full blast. He must get the pressure up on L'Heureux, never mind himself. Seventy pounds, with the valve wide open, the needle still racing up the dial. Eighty pounds, a higher pressure by far than anybody on that diving job had ever before been subjected to, either on the bottom or in the tank, and, worst of all for Flotte, taken in one swift rush!

Eighty pounds (55 m equivalent depth) was enough. Flotte shut off the air. Dizzy from the sudden impact of high pressure, ears ringing excruciatingly, he bent over L'Heureux, tore off his shirt. The diver's chest was covered with purple splotches, the result of the bursting of a myriad small blood vessels from expanding air. But that was a minor result of "the bends." The major question was circulation. Had he got those heart bubbles down before L'Heureux's heart had stopped forever?

Flotte bent over his chest, listened, then smiled wanly. His heroic treatment had succeeded. A faint heartbeat became perceptible, L'Heureux began to breathe again. The bubbles, compressed to one-sixth their previous size by the sudden application of

pressure, were passing out of the heart; blood was beginning to pump through it once more.

Gradually then Flotte began to release the air from the chamber, decompressing L'Heureux by regular stages. But in spite of working over him all night through, in spite of everything that his medical skill could suggest, Flotte was never able to bring L'Heureux back to consciousness. Through the long hours he lay there as the air pressure went down, limp, unconscious, apparently paralyzed in some degree, simply breathing feebly.

At 3 A.M., Dr. Flotte emerged from the recompression chamber, weak and dazed from his own exertions and the shock of high pressure. He sought out Lieutenant Hartley, skipper of the *Falcon*.

"Everything that pressure can do for L'Heureux's been done. Everything that I can do for him here has been done. He's paralyzed and he's nearly gone. If we're going to save L'Heureux's life, we've got to get him to a hospital right away!"…At 7 A.M. in the early dawn, we transferred the still unconscious L'Heureux to the ambulance and sadly headed back to sea...

That was mid-November. When we landed him, L'Heureux had been a man of something over 160 pounds weight. Within a few weeks, partial paralysis, including his kidneys, resulting from "the bends," had wasted him away to a skeleton of 70 pounds, and there for months he hovered precariously between life and death. Not until late the following July, after an eight-month struggle in the hospital, did he finally recover sufficiently to be discharged.

As an example of a severe case of decompression illness, probably with severe hypotension, one can only speculate as to the outcome that might have been achieved if the doctor had the tools available to measure blood pressure and administer intravenous fluids.

In fact, information about adjunctive treatment was recorded many years earlier, both by Alphonse Jaminet, physician responsible for the men constructing the bridge across the Mississippi River at St. Louis, beginning in 1868, and Andrew Smith, Surgeon to the New York Bridge Company during construction of the Brooklyn Bridge, in 1872. For the treatment of bends Jaminet recommended whiskey or beef broth.[2] Smith's recommendations included ergot by mouth or hypodermically, morphine or atropine for pain, friction with or without stimulating linaments, local hot water baths, an alcoholic stimulant, with ginger, for epigastric pain. For paralysis, he recommended cold douches and frictions to the spine, cups or leeches. Venesection was a possible recommendation for coma.[3-5] The efficacy of these treatments remains unreported.

Current treatment

Although it is commonly assumed that decompression illness (DCI) is a disease that is easily treatable, at least in recreational divers, treatment is considerably less than uniformly successful. Statistics from the Divers Alert Network indicate that after completion of a course of hyperbaric oxygen therapy one third of divers have residual symptoms.[6] The challenge is to find ways to improve the prognosis for those divers, especially ones with neurological symptoms who cannot receive immediate recompression with oxygen, who in general respond less well to recompression treatment.

Accepted modern day adjunctive therapy may consist of surface oxygen, fluid resuscitation, management of plasma glucose, corticosteroids, anticoagulants and management of core temperature.

SURFACE OXYGEN

Oxygen delivery kits for divers are widely available. The Divers Alert Network (DAN) sells a number of models of a demand flow apparatus and a rebreather kit. Other rebreathing systems are available in Australia and Switzerland.[7,8] The rationale for administration of oxygen on the surface is that by excluding nitrogen from the inspired gas the tissue blood PN_2, and then tissue PN_2 are reduced and the gradient for diffusion of gas out of the bubble into the blood increased. For patients who are hypoxaemic, due to aspiration or pulmonary barotrauma, correction of the low PO_2 is another benefit.

Evidence that surface oxygen works was provided by Dr Annane and colleagues,[9] who injected air into the carotid arteries of dogs until bubbles could be seen on a CT scan of the brain. They then compared the rate of resolution of cerebral air under two conditions: spontaneous breathing with room air and mechanical ventilation with 100% oxygen. CT scans of the brain were obtained every minute and revealed that bubbles resolved more quickly under the latter condition.

Data from the Divers Alert Network (DAN) also supports the benefit of surface oxygen (see Figures 1 and 2). While present evidence suggests a beneficial effect before recompression, it is not yet confirmed that ultimate outcome is improved.

FLUID RESUSCITATION

Dr Jaminet measured the specific gravity of urine in St Louis Bridge caisson workers. From his observations it can be discerned that workers who had symptoms of DCI had higher urine specific gravity than those who had no symptoms,[2] from which it reasonable to hypothesize that

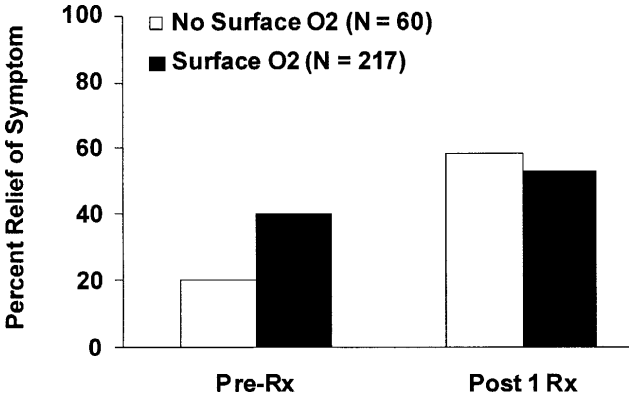

Figure 1. Percentage of divers with difficulty walking reporting complete relief before and after a single recompression treatment (from Divers Alert Network). Surface O_2 administration is associated with relief of symptoms prior to recompression (P = 0.003).

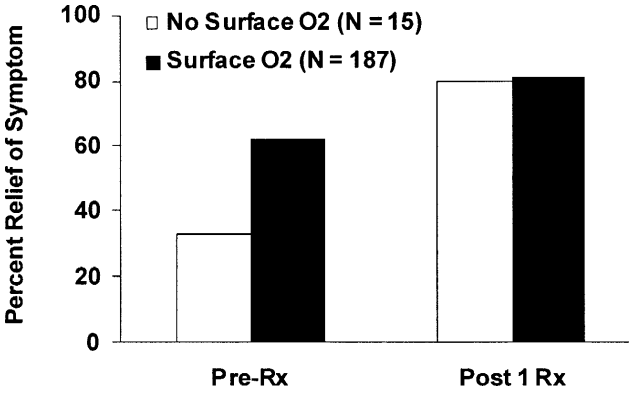

Figure 2. Percentage of divers with altered consciousness reporting complete relief before and after a single recompression treatment (from Divers Alert Network). Surface O_2 administration is associated with relief of symptoms prior to recompression (P = 0.029).

dehydration was either a result of bends or a predisposition to it.

Since then investigators in Zurich published a report describing two experimental divers with severe bends, in whom plasma volume was formally measured using a radioactive tracer. In these cases the venous haematocrit was close to 70% and the measured plasma volume significantly below normal, implicating plasma can leak into the interstitial space.[10] A study by Dr Boussuges and colleagues in France examined outcome after treatment of DCI as a function of initial haematocrit.[11] The investigators observed that those who had neurological sequelae, had a significantly higher haematocrit at presentation compared with those who recovered. These clinical data provide strong circumstantial evidence that fluid resuscitation is beneficial for severe bends.

There is indirect evidence that aggressive hydration can result in more rapid elimination of anesthetic gases,[12] suggesting that a similar approach in divers with decompression illness may accelerate the washout of excess inert gas. Indeed, interventions which increase central blood volume and cardiac preload, and hence raise cardiac output, such as supine position and head down tilt significantly increase the rate of inert gas washout.[13,14] Fluid administration may therefore be advantageous even in divers who are not dehydrated.

The most appropriate intravenous fluids for resuscitation of patients with DCI are either isotonic crystalloids (e.g. Ringer's solution, normal saline) or colloids. Rapid administration of hypotonic fluids to patients with injured brains can contribute to cerebral edema;[15] glucose-containing IV fluids could worsen neural injury (see below). Hypertonic saline (7.2%; 8 x normal) has been used in head injured patients with some success in reducing cerebral edema,[16] but has not been tested in patients with DCI.

For mild bends oral fluids may suffice. Rehydration after oral fluid administration is related to the rate of transport of water and electrolytes across the intestinal mucosa and the rate at which ingested fluid is delivered to the intestine. The gastric distention that occurs after oral fluid intake stimulates gastric emptying. However, the gastric emptying rate may be reduced by protein, or glucose concentrations greater than 5% (252 mOsm/kg). Maximum water absorption occurs at a sodium concentration of 60 mM and glucose concentration in the range of 80-120 mM. While most commercially available soft drinks and juices have an osmolality higher than plasma, water absorption is greater when osmolality is low.[17] An ideal solution for rehydration in diarrhea (possibly approximating the requirement in DCI) has been suggested as containing approximately 30-60 mM sodium, 70-150 mM glucose and osmolality of around 240 mOsm/kg.[18,19]

Plain water is almost always available, though its ingestion stimulates a urine output that is disproportionate to the degree of rehydration. Ingestion of electrolyte-free water causes a decrease in plasma osmolality and inhibition of ADH secretion. Urine output is then increased in response to the hypo-osmolality, and thus does not necessarily indicate adequate rehydration.[20,21] Studies of rehydration in normal volunteers in whom dehydration of 4% of body weight (12% reduction in plasma volume) was induced by exposure to a hot, dry environment indicate that administration over four hours of fluid equal to the volume lost, using water, failed to normalize plasma volume, although urine output had increased to 180-380 ml/hour.[20] Even after an additional 24 hours of ad lib fluid intake plasma volumes were 2.4-5.5% below pre-test values.

Although almost all commercially available beverages are low in sodium and high in carbohydrate, some

drinks marketed as "sports drinks" contain glucose and electrolytes that are close to ideal. If this is not available, a reasonable palatable oral rehydration fluid with appropriate electrolyte and carbohydrate concentration can be improvised by mixing one part orange or apple juice with two parts water and adding half a teaspoonful of salt to one liter of the mixture. Alternatively, in lieu of adding salt, one part sea water diluted with 9 parts fresh water can be used to dilute the juice. Provided the patient is not vomiting, an intake of 1,000-2,000 ml per hour for 1-2 hours is safe and usually well tolerated.

End points for fluid therapy should at least include normal hemodynamics and hematocrit. Urine output should exceed 1 ml/kg per hour, keeping in mind that if large volumes of hypotonic oral fluids are used, the urine output may falsely reflect the degree to which plasma volume repletion has occurred. Fluid should not be withheld just because an ideal liquid is not available.

HYPERGLYCAEMIA

There is evidence that hyperglycaemia can worsen central nervous system injury in both brain[22] and spinal cord,[23,24] probably due to accelerated production of lactate, and the ensuing intracellular acidosis. The effect probably becomes significant above a threshold plasma glucose of around 200 mg/dl (11 mM).[25,26] Administration of even small amounts of glucose, for example one litre of intravenous 5% dextrose solution, may worsen neurological outcome, even without significant hyperglycemia.[27,28]

Further evidence is available from a recent study of middle cerebral artery occlusion, in which both PO_2 and glucose were manipulated after inducing focal cerebral ischaemia of the parietal cortex of rabbits by cauterization of the right middle cerebral artery.[29] Serum glucose was varied between 2.8 mM and >28 mM; arterial PO_2 was either 50 mmHg or 150 mmHg. During hyperglycaemia, intracellular pH was reduced, mitochondrial function assessed by NADH redox state was impaired and infarct volume was greater than during hypoglycaemia.

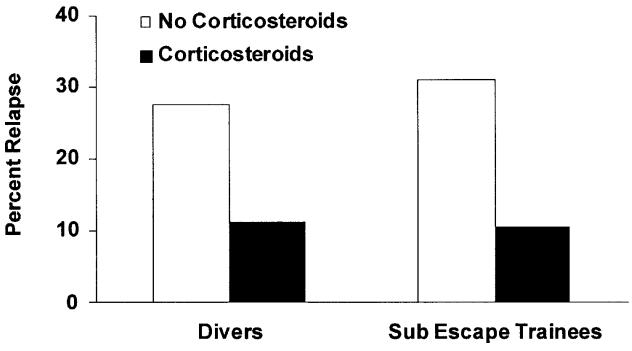

Figure 3. Relapse after arterial gas embolism as a function of whether corticosteroids were administered (from a retrospective study by Pearson and Goad).[36] Corticosteroid administration was associated with a significantly lower probability of relapse (P = 0.02).

Unless it is necessary to treat hypoglycemia, it is best to avoid the administration of glucose-containing intravenous solutions, and to measure plasma glucose if there is reason to suspect hyperglycaemia (e.g. if high dose corticosteroids are administered). Although there are relatively few diabetic divers, in glucose intolerant individuals stress and administration of corticosteroids can cause significant elevation of plasma glucose, which may require treatment.

MANAGEMENT OF CORE TEMPERATURE

Studies in which body temperature has been manipulated shortly after brain injury have shown that hypothermia can be beneficial, while hyperthermia is detrimental.[30-32] In a recent study patients with severe head injury (Glasgow coma scale 3-7) were randomly assigned to receive standard therapy or to be cooled to 33°C, kept at 32-33°C for 24 hours and then rewarmed. At 3 and 6 months after injury, for the patients with scores of 5 to 7, hypothermia was associated with significantly improved outcomes. The facility to induce and manage hypothermia is not widely available, and the observations need to be repeated before introducing the technique into routine practice. Thus, while diving doctors should not yet feel compelled to make divers with neurological injuries cold, it is worth a vigorous effort to be sure they do not become febrile.

CORTICOSTEROIDS

Steroids have been tried in most neural injuries, including DCI, and there have been several studies in animals. In a canine preparation, dexamethasone 1 mg/kg given before embolisation appeared to have a therapeutic effect upon the somatosensory evoked response amplitude, but when given afterwards there was no significant effect. Dr James Francis and colleagues, using methylprednisolone 20 mg/kg and recompression in spinal cord decompression sickness in dogs, observed no beneficial effect on somatosensory evoked responses within 250 minutes after treatment.[33]

Since then a study in patients with spinal cord trauma showed that methylprednisolone 30 mg/kg as a bolus, followed by 5.4 mg/kg/hour for 23 hours, was associated with greater recovery of motor function after 6 months.[34] In another study all patients received a bolus of methylprednisolone 30 mg/kg.[35] They were then randomized to receive methylprednisolone 5.4 mg/kg per

hour for either 24 or 48 hours. A third group received tirilazad mesylate 2.5 mg/kg every 6 hours for 48 hours. Patients treated within 3 hours of injury had equivalent outcomes at 6 weeks and 6 months. For those in whom therapy was initiated between 3 and 8 hours the patients treated with methylprednisolone for 48 hours did best, and those treated for 24 hours did least well.

Drs Pearson and Goad, in 1982, performed a retrospective study of relapse after arterial gas embolism, and reported a significantly lower incidence of relapse in patients who received one dose or more of corticosteroids (Figure 3).[36]

Whether corticosteroids should be administered routinely to divers with neurological DCI therefore remains undecided. On the basis of the information from studies in spinal cord trauma, if corticosteroids are administered they should probably be given early, preferably within the first 8 hours after symptom onset.

ANTICOAGULANTS

Evidence exists that bubbles in blood can initiate platelet adhesion and activation.[37-41] Conditions that could promote platelet aggregation might include large volumes of intravascular bubbles and sluggish blood flow, such as in the epidural venous plexus in severe decompression sickness.[42,43] Vascular obstruction due to bubbles could then be compounded by formation of fibrin clot. Anticoagulation of patients with neurological decompression illness might therefore seem reasonable.

However, in a study of arterial gas embolism in dogs,[44] in which somatosensory amplitude was the end-point, prostaglandin I_2 (PGI_2), indomethacin and heparin, and all possible combinations, were tested. None of the drugs singly or in double combinations were effective in returning somatosensory evoked potential amplitude toward normal. Only the combination of all three had any significant effect.

Anticoagulation may carry with it some risk. Tissue haemorrhage has been observed in animal and human decompression sickness of both the spinal cord and inner ear.[45-48] There has therefore been some reluctance to induce full heparinization in DCI. However, immobility due to spinal cord decompression illness is associated with deep vein thrombosis (DVT), and sometimes fatal pulmonary embolism.[49] Therefore, at least DVT prophylaxis is recommended for such patients.

The future

The initial event in decompression illness (DCI) is the formation of bubbles within tissue, which can cause immediate effects such as mechanical distortion and vascular occlusion leading to ischaemia. If reduction of oxygen delivery is extreme, rapid cellular death may occur due to depletion of ATP and loss of ionic membrane gradients. If, because the mechanical effects or ischaemia are less severe, immediate cell death does not ensue, then secondary injury mechanisms may ensue. Fibrin clot formation may occur, initiated by platelet activation. Ischaemia can initiate a cascade of events that includes excitatory neurotransmitter release, increase in intracellular calcium, sodium and zinc, increase in extracellular potassium, activation of proteases, adherence of leukocytes to endothelium in the ischaemic area, generation of reactive oxygen species such as superoxide and lipid peroxidation.[50] One of the effects of intracellular calcium influx is activation of the enzyme nitric oxide synthase, which catalyzes the formation of nitric oxide from arginine and oxygen. The resulting excess of nitric oxide (NO) can combine with superoxide anion (O_2^-), producing peroxynitrite ($ONOO^-$), a substance that can initiate DNA damage. This leads to activation of the nuclear repair enzyme poly(ADP-ribose) polymerase (PARP), which catalyzes attachment of ADP ribose units from NAD to nuclear proteins following DNA damage. This process consumes energy, and there is strong evidence that excessive activation of PARP after ischaemic cell injury can lead to cell death by energy depletion.[51] Compared with wild-type mice, animals with disruption of the gene that encodes PARP have 80% reduction in infarct volume after two hours of middle cerebral artery occlusion.[51] Evidence of PARP activation in the brain has been observed in humans after cardiac arrest,[52] and it has been suggested that PARP inhibitors could provide a potential therapy in acute stroke.[53] Indeed, treatment of rats with a PARP inhibitor significantly reduced the lesion volume that resulted from a 90 minute period of cerebral ischaemia.[54,55]

Clinical observation indicates that patients usually respond to treatment with hyperbaric oxygen hours, or even days after the insult, supporting the notion that immediate cellular death cannot be the only mechanism for DCI, and that there may be a “window of opportunity” in which adjunctive agents could limit secondary effects of bubbles pending definitive treatment with hyperbaric oxygen. Development of such agents should be a high priority for diving medicine. However, while in the USA per year there are approximately 1,000 cases of decompression illness, 1.5 million new cases of cerebrovascular disease occur. The vast weight of research effort is therefore directed toward the latter, and to the extent that DCI is due to ischaemia, solutions are likely to come from studies primarily directed toward treatment of stroke. Several potentially useful compounds may soon be available, as shown in Table 1.

LIGNOCAINE

Lignocaine is a promising agent of particular

TABLE 1

PARTIAL LIST OF ADJUNCTIVE AGENTS THAT HAVE BEEN INVESTIGATED IN CENTRAL NERVOUS SYSTEM ISCHAEMIA, TRAUMA OR DECOMPRESSION ILLNESS

(† indicates an absence of data in DCI.)

Compound	Mechanism	Animal Studies	Human Studies
Ancrod (viprinex)	Reduced fibrinogen	Neuroprotective in focal ischaemia[83]	Initial data favourable;[84,85] study ongoing †
Aspirin	Cyclooxygenase inhibition, anti-platelet effects		Anecdotal efficacy in DCI[86]
Clomethiazole	$GABA_A$ agonist	Neuroprotective in focal ischaemia and spinal cord injury[87,88]	Under investigation in stroke: initial report showed no effectiveness, although possibly effective in subgroups[89] †
Diaspirin cross-linked hemoglobin	Increased O_2 delivery	Neuroprotective in spinal cord ischaemia, cerebral ischaemia[90,91]	†
Doxycycline	Neutrophil inhibition	Improved outcome after AGE[92]	†
DPQ (3,4-dihydro-5-[4-(1-piperidinyl)butoxy] -1(2H)-isoquinolinone)	PARP inhibition	Neuroprotective in focal ischaemia[55]	†
Heparin and low MW heparin	Anticoagulation	Ineffective alone in AGE[44] but demonstrated to inhibit leukocyte rolling and sticking[93]	Possibly useful in preventing DVT in paraplegia due to DCI
Indomethacin	Cyclooxygenase inhibition, anti-platelet effects	Ineffective alone in AGE[44,] reduced pulmonary oedema due to venous gas embolism[94]	†
Isoproterenol	Increased intracellular cAMP	Reduced pulmonary oedema due to venous gas embolism[94]	†
Lignocaine	? inhibition of leukocyte activation	Improved outcome after AGE[56-58,94]	Reduced neurological deficit after CP bypass,[96] anecdotal improvement in DCI[61,62] and AGE[63]
Lubeluzole	Sodium channel blocker, inhibition of glutamate release	Neuroprotection after 9 minutes of bilateral and carotid artery occlusion hypotension in rats.[97] Lubeluzole and diaspirin cross-linked hemoglobin combination reduced infarct volume after focal cerebral ischaemia in rats[98]	Preliminary evidence supports effectiveness in human stroke[99] †

TABLE 1 (Continued)

PARTIAL LIST OF ADJUNCTIVE AGENTS THAT HAVE BEEN INVESTIGATED IN CENTRAL NERVOUS SYSTEM ISCHAEMIA, TRAUMA OR DECOMPRESSION ILLNESS

(† indicates an absence of data in DCI.)

Compound	Mechanism	Animal Studies	Human Studies
Methylprednisolone	? free radical scavenger	Effective in preventing paraplegia after aortic cross clamping (30 mg/kg before and after).[100] Acute study: 20 mg/kg ineffective in spinal cord DCS[33]	154 mg/kg over 23 h reduced 6 month morbidity in traumatic spinal cord injury[34] †
Mg^{2+}	NMDA, Ca^{2+} antagonist	Neuroprotection in spinal cord ischaemia[101,102]	†
Nicardipine	Inhibition of intracellular calcium influx, vasodilatation	Accelerated neurological recovery in dogs with hyperbaric oxygen after 5 min. global cerebral ischaemia[103]	†
Nifedipine	Inhibition of intracellular calcium influx, vasodilatation	Reduced pulmonary edema due to venous gas embolism[94]	†
Nimodipine	Inhibition of intracellular calcium influx, vasodilatation	Inconsistent effects after spinal cord injury[104-106]	Not useful in stroke[107] †
NMDA blockers	Prevention of Ca^{2+} entry	Neuroprotective in focal ischaemia[108,109]	Under investigation in stroke[110] †
Perfluorocarbons (e.g. Oxygent™)	Inert gas scavenger, improved O_2 delivery	Improved outcome after AGE, DCS[73,74,76,78, 80,82,111]	†
Tirilazad	Free radical scavenger	Neuroprotective in spinal cord injury[112-114]	Evidence of efficacy in traumatic spinal cord injury[35] †
Tissue plasminogen activator	Clot lysis		Effective in human stroke[115] †

interest because it is already on the market, and with both animal and human data suggesting a benefit. Dr Evans showed that anesthetized cats pretreated with lignocaine experienced less decrement in somatosensory evoked potential amplitude compared to control animals after vertebral artery embolization with air.[56] A beneficial effect was also observed when lignocaine was administered after embolization.[57] Dr Dutka and colleagues have also demonstrated that lignocaine-treated embolized dogs recover with significantly more SEP amplitude after recompression than dogs treated with recompression alone.[58] Lignocaine treatment attenuated the neurological injury produced by retrograde cerebral perfusion in dogs.[59] A randomized trial in humans by Dr Mitchell and colleagues demonstrating that lignocaine infusion improves outcome after cardiopulmonary bypass[60] and four cases of apparent benefit of lignocaine administration in decompression illness[61,62] and arterial gas embolism[63] support the animal data and provide a strong rationale for a study of lignocaine treatment in acute decompression illness in humans.

The mechanism of the apparent beneficial effect of lignocaine in this context is unknown, but may only be related to bubbles indirectly. Anecdotal evidence supports its use in ischaemic myelopathy due to scoliosis surgery.[64] High doses (160 mg/kg) may reduce the oxygen requirement of CNS tissue not only by inhibiting electrical activity, but also by blocking sodium and potassium ion leak fluxes.[65] Lignocaine (total 6 mg/kg) has been reported to reduce neurological damage in dogs after hypothermic circulatory arrest.[66] Lignocaine also inhibits the release of calcium from mitochondria into the cytosol during ischaemia,[67] and to attenuate extracellular glutamate accumulation.[68] Lignocaine has been observed to attenuate the acute lung injury produced by pancreatic enzymes.[69] A study demonstrating that intravenous lignocaine attenuates the pulmonary damage induced by aspiration of hydrochloric acid, mediated at least in part by inhibition, sequestration and activation of neutrophils, suggests that lignocaine may inhibit the elaboration of oxygen free radicals induced by gas embolism.[70]

Intravenous administration of lignocaine requires an infusion pump and the capability of dealing with untoward effects such as seizures. "Field" use of lignocaine, using injection of 200-400 mg into the deltoid muscle, produces plasma concentrations in the therapeutic range for arrhythmia prophylaxis for up to two hours.[71]

PERFLUOROCARBONS

Perfluorocarbons have an extremely high solubility for a variety of gases, including N_2 and O_2, providing a rationale for their use in DCI. Intravenous administration of perfluorocarbons could provide a gas trap to accelerate diffusion of nitrogen from bubbles into the blood, while simultaneously enhancing O_2 delivery. Intravenous pretreatment of animals with perfluorocarbons prior to intracarotid air injection is protective against neural injury and retinal injury.[72-76] Similar protection has been observed for cardiac damage after coronary air embolism and cardiorespiratory effects after venous gas embolism.[77,78] Inert gas elimination from muscle is enhanced by perfluorocarbons.[79] Rats with decompression illness treated with intravenous FC-43 and 100% O_2 survived significantly longer than controls receiving 6% hetastarch or saline.[80-82] A new perfluorocarbon (Oxygent™, Alliance Pharmaceutical Corp., San Diego, California, USA) is expected to be approved shortly by the American Food and Drug Administration. Testing in DCI will be needed to show efficacy in humans. It will also be important to demonstrate that it does not promote O_2 toxicity.

Summary

Surface oxygen appears to be efficacious, at least whenever there is some delay to recompression treatment. First aid measures should ideally include airway management and assuring adequacy of ventilation, fluid resuscitation and maintenance of blood pressure. Other principles include avoidance and treatment of hyperglycaemia and hyperthermia. Deep vein thrombosis prophylaxis is recommended for divers with severe leg weakness.

In the near future several pharmacological agents may become available to protect against neurological damage during the pre-recompression interval.

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AUDIENCE PARTICIPATION

John Knight, Melbourne.

As another anaesthetist, my only quibble with the positioning of the airway at the end of the list. I think the airway should be first, because it is the easiest one to do. Intravenous fluid requires skills that most divers do not have, and you do get people in the general community who can position people on their sides so that they can breathe again. I think we ought to be concentrating, when we talk about early treatment, not on what doctors can offer, but what the other people on the dive boat, the dive guides and the other divers, can offer.

Richard Moon.

Yes. I absolutely agree; the point is well taken and thank you.

Ian Seppelt, Sydney.

When you are advising non-medical people who are treating an accident, do you ask them to give air breaks, or do you say continuous surface oxygen as long as possible? How long do you accept if it is going to be a few hours until they get to you?

Richard Moon.

Since oxygen toxicity is unlikely during administration of surface O_2 for a few hours, I do not recommend scheduled air breaks.

Mike Loxton, Royal Australian Navy.

Just an observation on the surface oxygen. We did a retrospective study of about 260 cases treated at the School of Underwater Medicine from 1983 to 1993, and certainly we found in the univariate analysis a very significant advantage in using surface oxygen, but when we allowed for confounders, we did not find a statistically significant advantage. The main confounder appeared to be treatment delay. Those patients given surface oxygen were treated a lot earlier, and after allowing for that, we did not find a statistically significant advantage. Just an observation, and

perhaps the number of cases would not allow me not to advise surface oxygen.

Richard Moon.

Your point, that confounding variables may explain what appears to be a beneficial effect of surface oxygen, is a good one. For example, someone prepared with the kit necessary to administer surface oxygen might be more likely to transport an injured diver expeditiously to a treatment facility, and therefore obtain a better outcome because of speedy treatment. I think it is possible that our data underestimate the effect of surface oxygen, because it is often administered in a less than intensive manner. It is often given for only a few minutes at a time; in nearly a third of cases reported to DAN in 1998 it was administered for less than an hour.

Gordon Bentley, Brisbane.

Faced with a long journey and a limited supply of oxygen, would you recommend giving a high concentration for a short time, or a lower concentration throughout the entire journey?

Richard Moon.

That is an excellent question, to which I do not know the answer.

Fiona Sharp, Perth.

Tirilazad been shown to be a benefit in males for spinal cord injury and trauma. Have there been any studies done on tirilazad being used in decompression injuries involving the spinal cord?

Richard Moon.

As far as I know, it has not.

Tony Lee, Malaysia.

There used to be some interest in lignocaine. Has it gone numb?

Richard Moon.

Although there is experimental evidence, and some anecdotal clinical evidence, that lignocaine is effective in decompression illness, in this setting I would still consider it an experimental drug.

Brian Casey, Sydney.

You made reference to plasma in one of your studies in terms of intravenous fluid use. I wondered what particular intravenous fluids you would use if you had got everything available, plasma, Ringer's lactate, saline?

Richard Moon.

I support Alf Brubakk's point, that hypotonic fluids are not good choices. Whether isotonic colloid or crystalloid fluids are preferred is not yet established. In anesthetic practice in the US we tend to be concerned about cost, so in the absence of evidence in favor of using colloids, we tend to favor crystalloids. Whether there is any difference in effectiveness between Ringer's lactate and normal saline in DCI, I do not know, but I would suspect not.

Akin Toklu, Turkey.

Is there any experimental study which suggests not to use aspirin?

Richard Moon.

There are anecdotal stories about improvement after using aspirin in neurological bends, but I think we do not really know. Dr Mike Bennett in Sydney is doing a study on a non-steroidal anti-inflammatory drug, which may have a similar effect to aspirin, and I look forward to his presentation in a few months or a year to give us the answer on that.

Alf Brubakk.

I do not think there are any studies on aspirin. But one point is that the aggregation of thrombocytes that one sees caused by gas bubbles does not seem to be influenced by aspirin. It seems to be due to a different mechanism. Another point is that there are some older studies which seem to indicate that several non-steroidal anti-inflammatory drugs seem to have a dramatic effect on decompression sickness. There are several mice studies which have not been repeated for 30 or 40 years, which would be very interesting to repeat. There is other data to indicate that it might do some good. That is all I know about it.

There was a question about oxygen, how much should you use? Do you give a high concentration for a short time, or a lower concentration over a longer period? I think the answer depends on when you start to give it. Do you start giving it immediately? It is obvious that the higher the oxygen concentration, the faster the bubbles will shrink. If you are after an immediate treatment effect to get rid of the bubbles, then I would say use as much oxygen as you can, as early as you can. When it comes to secondary effects, it is much more complicated, and I cannot answer that.

Richard Moon.

It is interesting that patients will often tell you that they breathed oxygen for a while, and there was improvement, but when they stopped breathing it, the symptoms came back.

Chris Acott, Adelaide.

A couple of years ago I was looking up about oxygen, intravenous fluids, steroids, the French regime that Fructus invented, and aspirin. There were only 40 or 50 patients, not very many, but the ones who actually received aspirin as well as steroids, and oxygen, and fluids actually probably did a little bit worse that those who did not, in the long term.

Robyn Walker, Royal Australian Navy.

Getting back to oxygen and fluids. I think while we are telling everyone to use them, we have to encourage people to talk to the treating unit. Just this week, we received a patient, evacuated from Vanuatu by a private company. We were not asked to comment on how she should be transported. She had 15 hours of 100% oxygen without an air break. Although she arrived asymptomatic she had significant pulmonary oxygen toxicity. Those who arranged the evacuation had not realised that oxygen toxicity could be a problem for us if we then had to treat that person in the chamber. Secondly, she was given five litres of intravenous fluid in five hours. However, she was asymptomatic when that fluid treatment was commenced. Certainly while fluids and oxygen are very important, I think we need to give people some advice before patients are transported. I would urge them to talk to the treating unit before transport.

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DECOMPRESSION ILLNESS SEQUELAE IN TUNA FARM DIVERS

Chris Acott

Key Words

Decompression illness, hyperbaric oxygen, occupational diving, sequelae, training, treatment.

Introduction

Fisherman divers, the abalone divers of south and south-eastern Australia, salmon farm divers of Tasmania and the pearl divers of the tropical north, are part of the fishing culture of Australia. Some Australian coastal towns are dependent on such fishing.

Tuna fish are one of South Australia's natural resources. The majority of tuna fishing is conducted in the Southern Ocean by vessels based in Port Lincoln.

Port Lincoln, near the tip of the Eyre Peninsula, was first settled in 1834. It was to have been the capital of the new colony of South Australia because of its deep natural harbour. However, lack of an adequate fresh water supply and the low rainfall inland saw that Adelaide, 250 km (156 miles) east as the seagull flies, became the capital. Port Lincoln, with a population of 12,000 and over 600 km (375 miles) by road from Adelaide, is the centre of the South Australian Tuna industry. Its main industries are fishing (tuna and abalone), grain exporting, tourism and wine production.

Tuna fishing

Until the 1990s the Tuna industry used single vessel techniques, baited lines, often attached to poles which enabled strong men to swing the heavy fish inboard. Tuna schools are now co-operatively netted in the open sea and then the nets are towed back to Port Lincoln. Here the tuna are kept in netted enclosures, near the shore, for fattening before harvesting. The main export market for the tuna is Japan. Divers are employed for net maintenance, clearing the dead tuna from the enclosures and in the tuna harvest. In the early years harvesting was by swimming each fish to the surface, which involved many extremely rapid ascents, from depths of up to 18 m, in each "dive". This practice contributed to the high incidence of decompression illness (DCI) and was stopped by regulations introduced in 1995.[1]

The tuna are kept inside an inner net while an outer net prevents any intrusion by sharks. Sharks have been found between the nets but to date (May 1999) no diver has been attacked by a shark, however, this may reflect a lack of reporting of any such attack.

Government marine biologists have expressed concern about the impact the nets have on the environment. All the debris from feeding and fish excrement are deposited below the nets, and no attempt has been made to clear this rubbish away. Already one storm has stirred up this debris and suffocated millions of dollars worth of fish. Furthermore the presence of the tuna has lured sharks, in particular Great Whites, to the area where the nets are sited. These areas are close to the local beaches.

The divers

Between August 1993 and January 1995, 17 divers employed in the tuna industry were treated for decompression illness by the Hyperbaric Medicine Unit at the Royal Adelaide Hospital [RAH HMU]. Many of these 17 divers had continued to dive while symptomatic. In all but one case there was a delay before medical treatment was obtained. From January 1995 a further four divers have been treated, making a total, to May 1999, of 21 divers.

Amazingly, the initial response by the South Australian Government and Medicare, the Australian Federal Government's national health insurance system, to this cohort of 17 divers with DCS was that the RAH HMU's

medical practitioners were "over servicing" these divers and so were guilty of fraud. This was despite the RAH HMU's attempts to alert the appropriate South Australian regulatory body, the divers and their employers to the dangers of their diving practices. It was later discovered that an investigation into the treatment practice of the RAH HMU had been carried out secretly by Medicare. By the end of 1994 the bureaucrats finally recognised that there was a significant problem and regulations for safer diving practices were put in place, despite considerable opposition from both the tuna farm owners and the divers' employers.

From 1995 these regulations produced by the Department of Industrial Affairs and Workcover have resulted in a decrease in the incidence of **reported** cases of DCI. The actual incidence of decompression illness in this diving population is unknown. Many divers elect not to seek treatment nor to report their symptoms for various reasons, the main one being a fear of losing their employment. However there have been two deaths since the 1995 regulations were introduced. Despite regulations requiring training to occupational diver standards before employment as a tuna farm diver being in place, in 1996 an untrained diver failed to surface after running out of air. The other death was a suicide related to the death of the first diver. These two deaths are under police and coronial investigation and will not be discussed.

Diving practices

The initial working environment was undisciplined and unregulated. Diving profiles involved multiple rapid ascents, multiple dives per day and multiple days diving, with only one day off per week. Following their daily diving duties the divers were involved in hard manual labour.

The divers' diving experience ranged from ex-Royal Australian Naval divers to those who had recently obtained a recreational open water diving certificate. Frequently, Port Lincoln's unemployed youths were sent by the local government unemployment agency to the local dive shop to obtain a recreational diving qualification to enable them to be employed as a tuna farm diver. This not only decreased the area's unemployment figures but also gave the tuna farmers a cheap labour force.

The majority of the dives were done using surface supply, from petrol driven compressors which were frequently left unattended. Compressor malfunction, usually running out of petrol, inevitably caused an out of air ascent. Verbal communication systems and "bail out" bottles were not used.

Since 1995 all tuna divers must be trained to occupation air diving standards. The 1995 regulations, when adhered to and enforced, have altered diving practice for the better. However, there are still many instances where divers have run out of air due to compressor malfunction. Unfortunately, it appears that bail out bottles and a diver's attendant are only present when a "secret inspection" is known to be about to occur. In 1996 an untrained diver died while diving in the nets.

The tuna industry's diving practice will always differ substantially from other occupational, recreational or abalone diving practice. Indeed, its nature may be provocative for decompression illness but safe diving practice should never be discarded because of cost or convenience.

Treatment

Divers have been initially treated with a RN 62 with daily follow up treatments. The follow up treatment tables used were at the discretion of the physician in charge. Usually a 60 minute "soak" at 18 m, followed by a 30 minute ascent was used. This is the RAH HMU's 18:60:30 treatment table. Daily soaks were continued until the diver's symptoms had ceased improving.

The initial treatment response was good but subsequent follow up produced 9 divers with an unusual pattern of symptoms.

Post-treatment syndrome

Nine divers, three of whom have since been lost to follow up, developed a post-treatment syndrome characterised by:

1 generalised arthralgia, muscle and bone pain which is worse in the winter;
2 fatigue and weakness;
3 agitated depression;
4 mood swings;
5 poor libido;
6 breakdown of personal relationships;
7 and cognitive dysfunction, poor short term memory and concentration problems.

All of these symptoms and signs have been exhibited by the majority of these 9 divers at some stage after treatment. The muscle and joint pain is crippling at some stages during the winter. Four of the divers have developed degenerative changes to their right acromio-clavicular joint and two have symptoms of prostatism.

All investigations and specialist consultations have been inconclusive. Bone scans, plain X-rays and MRI have been negative for osteonecrosis. Some bone scans have been positive but the follow up MRIs have been negative. In one diver the MRI scan was positive but subsequent screening was negative. Rheumatological investigations

have all been negative for any arthritic conditions. Two divers have undergone arthroscopies, synovial fluid analysis and synovial biopsies of their worst effected joint, these have all been negative. The RAH's Chronic Pain Unit has had little success in controlling their pain. Urological consultation has not shown an enlarged prostate but has demonstrated poor bladder capacity.

Psychiatric consultation has helped in diminishing their depression and anger.

Their rehabilitation has been hampered by:

1 an inability to move fluently,
2 feelings of anger and resentment directed particularly towards their employers and Workcover,
3 lack of insight into their problems,
4 inability to accept that their injury did not respond well to treatment
5 and a lack of understanding and support by their local community and peers.

In many respects their complaints are similar to those reported by other Australian and New Zealand hyperbaric units which have reported their DCI cases after follow up for a year or more. The earliest report was by Gorman et al. in 1987.[2] They reported on patients treated by the Royal Australian Navy in Sydney. Those who had longstanding sequelae largely had EEG and neuropsychiatric changes. In 1989 Sutherland found that of 30 cases, 23 could be followed up a year later. Of these 8 were permanently and significantly damaged as a result of their diving accident and five were unable to return to normal employment.[3] Sutherland, Veale and Gorman reported in 1993 that 74% of their patients were left with problems.[4] The complaints, in order of frequency, were mood disorders (including mood changes from uncontrollable irritability to depression, lassitude and social withdrawal), impaired short term memory and often other problems such as difficulties with arithmetic, headache, sensory disturbances, impaired balance, motor weakness, arthralgia and myalgia, visual disturbances, dysphasia and dyslexia, bowel and bladder problems.

About the same time Sutherland reported a patient who had suffered personality changes and difficulty with his hands. These led to writing problems and difficulty with employment.[5] The most recent report is from Chapman-Smith whose patient developed psychosexual dysfunction, hands seizing up and short term memory problems.[6]

Although some of the patients in these reports reported arthralgia and myalgia there have been no earlier reports of muscle and bone pain which gets worse in winter nor of the development of degenerative changes in the joints.

Discussion

Divers suffering from this post-treatment syndrome present a management problem which does not seem to have a solution. The majority of our affected divers are seeking compensation. It will be interesting to see if these symptoms persist when compensation is paid.

The future

A longitudinal survey of the health of tuna farm divers in South Australia, funded by Workcover and the Department of Industrial Affairs, is being conducted. The principle researcher is David Doolette. The study is to define the true risk of decompression illness in the tuna farm divers and the risk factors associated with diving in the aquaculture industry. The aim is to improve the health of tuna farm divers and to prevent a similar scenario being repeated in other aspects of the emerging aquaculture industry in South Australia.

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

REVERSE DIVE PROFILES WORKSHOP
Washington DC. October 29-30, 1999

R W Hamilton and Erik Baker

Key Words
Decompression illness, diving theory, physiology, tables.

For many years recreational scuba divers and their instructors have embraced a maxim that it is unacceptable to perform a repetitive dive to a deeper depth than a previous dive, or to perform the deeper part of a single multilevel dive after a shallower part. These two manifestations of the same dive pattern are known (by some) as "reverse dive profiles." While there is widespread recognition among recreational divers that reverse dive profiles are forbidden, such profiles are apparently being performed anyway. It is not so clear where the rule against reverse dive profiles came from or how significant it really is.

In recent years the recreational diver has seen the development and refinement of dive computers and "do-it-yourself" decompression computational programs for personal computers. Most of these do not specifically prohibit the pattern of reverse dive profiles and will allow the calculation of such profiles without apology. The primary consequence is that there may be less no-stop bottom time available on a repetitive dive. In professional practice, neither the military nor commercial diving communities prohibit or even recognise reverse dive profiles as unique, except in rare instances. These facts have recently called into question the long-standing prohibition against reverse dive profiles in the recreational diving community.

Since there are incentives of greater allowable bottom time for the recreational diver to observe the prohibition of reverse order and perform dives only in "forward" order, the rule has not been subject to serious questioning in the past. However, for scientific divers who mainly use the same techniques as recreational divers but who may have more demanding mission objectives for their dives, to follow the rule can impose a significant operational limitation. It seemed worthwhile to re-examine the rationale for this rule. This was recently done in a high-level international Workshop sponsored by the Smithsonian Institution and some other interested organisations and held 1999 October 29 and 30 at Smithsonian facilities in and near Washington DC.

The Workshop was organised by Michael Lang, Diving Officer and head of the Smithsonian Institution's Scientific Diving Program, and by Charles Lehner, Diving Physiology Laboratory, University of Wisconsin, Madison. Michael is not new to workshops on challenging subjects, having organised earlier workshops on dive computers, biomechanics of ascent rates, polar diving, and repetitive diving, all under the auspices of the American Academy of Underwater Sciences. In addition to the Smithsonian and AAUS, other sponsors of this Workshop include DAN, DEMA, and Dive Training Magazine.

Before even touching on the technical aspects of the Workshop, one truly unique aspect of it should be mentioned, the high level of talent that was assembled to tackle the issue of reverse dive profiles. The participants included most of the decompression modellers and theorists in current practice, and many practical developers of decompression tables, hardware, and software, military and commercial diving operational managers, diving training people and organisations, media representatives, manufacturers, and an impressive array of medical experts who see the results of decompression practices when they do not work quite right.

Participants making formal presentations (with co-authors who were not there in parentheses) in order of their appearance included: Michael Lang, John Lewis, Glen Egstrom, David Yount, (Eric Maiken), Erik Baker, Bruce Wienke, (Tim O'Leary), Hugh Van Liew, Peter Tikuisis, Ron Nishi, Charles Lehner, Valerie Flook, Alf Brubakk, (Olav Eftedal), Paul Weathersby, Wayne Gerth, Michael Gernhardt, (Ed Thalmann), Dick Vann, Petar Denoble, Karl Huggins, Till Mutzbauer, Bill Hamilton, Jon Hardy, Peter Mueller, Terry Overland, Drew Richardson, Karl Shreeves, (Jed Livingstone), Duke Scott, Ted Maney, Steve Sellers, Walter Jaap, Richard Moon and Tom Neuman. Those attending the Workshop and taking part in the discussions, but without formal presentations, represented an equally impressive array of expertise.

The Workshop's objective was to examine whether reverse dive profiles are cause for increased risk. To see if there was a real reason to prohibit such profiles, the issue was addressed along two lines—analysis of existing diving data and evaluation by decompression modelling.

The first session defined the issue of reverse dive profiles and included speculation on just where the "prohibition" against them originated. Prior to the Workshop some of the participants were not familiar with the term "reverse dive profile." Review of the literature in search of the origin of the prohibition suggests that the rule probably had less to do with safety issues and more to do with "optimising" bottom time over a series of dives. This comes from gas loading considerations that allow more useable bottom time by doing the deep dive first.

The Workshop accepted the definition of a reverse dive profile, but one definition that was not quite agreed upon was that of a "bounce dive." To recreationally oriented divers this usually means going straight down and coming straight back up, without decompression stops, a pattern resembling a "spike." In commercial and military diving, any dive not involving decompression from saturation would usually be regarded as a bounce dive, even if it involved a long decompression. Thus, the term "bounce dive" should be interpreted in context.

The next two sessions concentrated on physics, physiology, and modelling. Several of the latest decompression models were employed to analyse selected series of reverse dive profiles. Among the modelling approaches, bubble formation and/or growth models were prevalent. Although there was diversity between the bubble models, they tended to arrive at some similar conclusions. For example, most call for lower allowable supersaturation gradients on the initial stops ("deep stops") and shorter no-decompression limits than conventional dissolved gas models. The bubble models included David Yount's varying permeability model (VPM) also known as the "tiny bubble" model, Bruce Wienke's reduced gradient bubble model (RGBM), the Duke University bubble volume model, the DCIEM bubble evolution model based on Doppler scores, a gas dynamics model by Valerie Flook based on Van Liew's concepts, and Michael Gernhardt's tissue bubble dynamics model.

In his presentation, Hugh Van Liew made the argument that direct experimental validation is needed about the existence and role of micronuclei for bubble formation in mammalian tissues. This includes whether or not such gas nuclei can be "crushed" to the point of elimination or inactivation. In another presentation it was shown that the reverse dive profile may have a higher predicted incidence of DCS, but for pairs of no-stop dives the differences were trivial and a decompression using the US Navy tables would be adequate. However, for dives involving decompression stops or for more than two dives in a row, it looked like these tables might not provide a reliable decompression. All of this pointed toward an urgent requirement for more information, and to this end Alf Brubakk suggested an animal model that might at least show which profiles result in the most bubbles.

Another session included a panel discussion by several dive computer manufacturers. Many of the older computers on the market use conventional dissolved gas (Haldanian) algorithms that take into account only gas loading and supersaturation limits (M-values), and do not specifically consider the order in which dives are conducted. In these cases, the user manuals accompanying the computers may recommend against reverse dive profiles. Some of the latest dive computers incorporate algorithms that are based to varying degrees on bubble models; these computers have specific warning features or penalties for dive patterns associated with increased risk (spike, yo-yo, repetitive dives with excessive pressure differentials, etc.).

Regarding data, many horror stories have been associated with reverse profiles, the classical one being the instructor making a short, deep dive to release the anchor chain after a day of diving and getting severe DCS. This is hard to interpret because it is a very small "n", there is usually no denominator, and buddy divers doing the same profile may be unaffected. Other data showing say 100 dives may be insufficient for statistical analysis, but one comment put this into perspective: "We are a better off having that 100 dives than no observations at all." A number of participants reviewed some substantial data collections, including the US Navy, commercial diving, chamber data, DAN records, various sets of recreational dive data, and some significant contributions from field experience.

An argument can be made that the present lack of data that says reverse profiles are dangerous could be, in part, due to the arbitrary prohibition against them that has been in place for many years, so not so many of these dives have been done.

Although there were some problems with reverse dive profiles in isolated examples, the conclusion drawn from overall analysis of the actual diving data was that reverse profiles *per se* have not shown a higher risk for DCS than forward profiles. However, this holds most confidently when the differential pressure for the reverse profile is not too great, but this also means that depth is a factor, since you cannot get big differentials without having significant depth. It appears that decompression tables, algorithms and dive computers are adequately handling the issue of reverse dive profiles in the field.

Another observation is that this subject seems to be very much a matter of repetitive diving, and in general, this is handled quite differently across the many decompression algorithms.

After all the presentations were complete, Richard Moon and Tom Neuman provided "individual perspective" summaries of the information that had been presented at this Workshop. The discussion was then turned to the floor for purposes of arriving at a list of Findings and a Conclusion for the Workshop.

The discussion got a little heated when it came time to come up with a Conclusion or Recommendations. Several of the folks who work with bubble models had serious reservations about a "complete retraction" of warnings against doing reverse dive profiles.

In other words, the bubble models suggest that you might really get into trouble on an improperly planned or executed reverse dive profile. Many were concerned that divers, especially inexperienced sport divers, would get the

wrong message about reverse profiles and think that it was okay to do them without any special consideration.

A couple of key concessions were obtained by the bubble modellers. It was pointed out that practical diving experience showed that there had not been many problems with reverse profiles, but bubble models showed that there could be. So, some wording was adjusted to make it clear that it was only in the diving experience that there were few problems, not that there is a lack of evidence of any kind that reverse profiles are or could have a higher DCS risk. The sentiment prevailed also that there should be a pressure differential limit, or "delta-P," noting that most of the safely executed reverse profiles were 12 msw (40 fsw) or less between the repetitive dives. Another point of agreement was that the sport diving limit of 40 msw or 130 fsw should apply to any relaxation of current prohibitions on reverse profile diving.

The Workshop Findings

* Historically neither the US Navy nor the commercial sector have prohibited reverse dive profiles.
* Reverse dive profiles are being performed in recreational, scientific, commercial, and military diving.
* The prohibition of reverse dive profiles by recreational training organisations cannot be traced to any definite diving experience that indicates an increased risk of DCS.
* No convincing evidence was presented that reverse dive profiles within the no-decompression limits lead to a measurable increase in the risk of DCS.

Conclusion

The Workshop found no reason for the diving communities to prohibit reverse dive profiles for no-decompression dives less than 40 msw (130 fsw) and depth differentials less than 12 msw (40 fsw).

The proceedings have been published (295 pages in soft cover).

Lang MA and Lehner CE. Eds. Proceedings of the Reverse Dive Profiles Workshop. Washington DC: Smithsonian Institution, 2000

The proceedings are available from AAUS, 430 Nahant Road, Nahant, MA 01908. (781) 581-7370 x334. aaus@neu.edu. The retail price is $25.00. They are also available from Best Publishing Co., DAN and UHMS.

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RISKS REPORT SHEDS NEW LIGHT ON DEATHS

Anon

Key words
Accidents, deaths, incidents, risk.

The biggest causes of death among sport divers are entrapment and entanglement, air embolisms and reckless deep dives. And, directly related to embolisms, rapid ascents are the single most common type of diving incident.

These and other fascinating incident patterns have emerged with the publication of an in-depth study by the Health & Safety Executive (HSE).

The report *Scuba Diving: A Quantitative Risk Assessment* has been prepared by the Paras research group to quantify risks to diving scientists.

However, as few statistics exist for scientific diving, the report is actually based on sport diving information provided by the British Sub-Aqua Club (BSAC) and by the Divers Alert Network (DAN).

The BSAC provided information on 849 incidents from 1990 to 1994, of which 57 were fatalities; and DAN provided data on 277 fatalities to US citizens from 1992 to 1994.

A number of cases were excluded for various reasons including 48 fatalities of a cardiovascular nature, because the study was required to assess divers who would almost certainly have passed a diving medical.

Total analyses therefore covered about 1,000 incidents, of which 286 were fatalities.

The researchers broke cases down into types, including entrapment in closed environments, entanglement, rapid ascents and air embolisms, reckless diving, solo diving, states of health and loss of consciousness for unknown reasons.

They then broke these areas down further to establish causal patterns, which make for illuminating reading.

The most frequent principal causes of fatalities were entanglement and entrapment, air embolism and reckless deep dives.

Curiously, loss of consciousness was the next most frequent principal cause of death, research indicating that many divers who could have saved themselves following a serious incident failed to do so and drowned.

The report suggested that there may be a link between extreme fright and loss of consciousness. Key among contributory causes to fatalities was buddy separation. Failure to monitor air supply and inadequate dive briefings came next.

Health factors as possible contributory causes accounted for 2% of BSAC fatalities and 5.1% of DAN fatalities. Conditions encountered included obesity, diabetes, alcoholic effects, meditations, previous surgery, recreational drugs, asthma, AIDS, migraines and ovarian cysts.

The most frequent contributors to incidents, fatal or not, were rapid ascent; bad interaction between diver and boat; bad monitoring of time, depth and air supply; buddy separation; and inadequate dive briefing.

Rapid ascents, however, accounted for fewer breath-hold embolisms than might be thought. About half of all embolisms occurred during normal ascents, indicating that better diver education is required. It is known, for instance, that divers can underestimate the rapid pressure changes that occur in the last 10 m of an ascent.

A major lesson to emerge from the study was that many cases involve not one but two or more causal effects, which build up in a chain reaction to cause the final incident.

No fewer than 93% of the fatal incidents recorded involved multiple contributory causes.

By inference, divers who can recognise small but potentially dangerous problems and take steps to cover for them are much more likely to avoid harmful incidents under water.

Based on projections of numbers of divers, diving hours completed and fatalities, it was calculated that sport diving suffers roughly one death per 5,000 divers per year typical, stated the report, for adventure sports.

A copy of the report, immensely thorough with some case details, is available at £35 from: HSE Books, PO Box 1999, Sudbury, Suffolk CO10 6FS, United Kingdom (Phone +44-1787-881-165. Fax +44-1787-313-995).

Reprinted, by kind permission of the Editor, from DIVER, then the magazine of the British Sub-Aqua Club, 1998; 43 (1) January: 45

DIVER is published by Eaton Publications, 55 High Street, Teddington, Middlesex TW11 8HA, United Kingdom. The annual subscription is £ 33.00 (UK) £ 43 world wide surface mail. Payment may be by Visa, Eurocard, MasterCard or by sterling cheque (in English pounds).

TOO MANY RAPID ASCENTS GET A GRIP, SAYS BSAC

Key words
Accidents, deaths, decompression illness, incidents.

More divers are suffering from rapid ascents due to poor control over their drysuits and BCs. Yet at the same time, overall numbers of incidents resulting in decompression illness are down. And the 17 UK fatalities listed for 1999 represents a drop on 1998 (at 22, a particularly bad year), close to the average over the past five years (16.8), but a rise on the ten-year average of 14.8.

These and many more statistics, plus the incident stories behind the figures, are published in the annual *Diving Incidents Report* compiled by the National Diving Committee of the British Sub-Aqua Club (NDC).

Listing incidents reported by BSAC members and other divers in the UK, and BSAC members overseas, the report provides pointers on the causes of sport-diving accidents.

Of 382 incidents reported, 86 involved DCI, with 98 casualties, down on the 120 casualties for 1998. But the report states that "it is very likely that there are further cases of DCI". Uncontrolled ascents accounted for a higher proportion of incidents than before, indicating, says the report, a need for better training in the use of modern drysuit and BC systems.

Among problems leading to DCI, 31 per cent involved depths greater than 30 m, 29 per cent rapid ascents. 21 per cent miscalculated repeat diving, and 14 per cent missed decompression stops.

A study of depths at which all the in-water incidents commenced showed that the ratio of fatal incidents was "significantly higher" at depths of more than 50 m indicating that the potential for problems increased markedly with depth and backing the BSAC's own stipulation that its members should not exceed 50 m.

The number of incidents involving diver separation from boats leapt from 34 in 1998 to 51 for 1999.

There were 50 cases of boat engine failure and 13 incidences of other boat problems. This bears out the Coastguard's long-standing message that divers need to improve their boating skills and take better measures to ensure boat-todiver contact. If separation does occur, divers need to have effective location devices.

Reflecting on these other facts in the report, compiler Brian Cumming concluded, that: "Most of the incidents... could have been avoided had those involved followed a few basic principles of safe diving practice."

He implored divers to dive within their limits, building experience gradually; to practise basic skills until they were "faultless"; take dive planning and equipment preparation seriously; remain vigilant and ready to deal with setbacks while on a dive; and to ensure that both diving and boat equipment were well serviced.

A copy of the NDC *Diving Incidents Report* can be ordered from BSAC Headquarters.

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DIVER is published by Eaton Publications, 55 High Street, Teddington, Middlesex TW11 8HA, United Kingdom. The annual subscription is £ 46.00 (overseas surface mail) which may be paid by credit card.

GLEANINGS FROM MEDICAL JOURNALS

Defibrillation in Hyperbaric Chambers: a review
Pitkin A.
J Royal Naval Medical Service 1999; 85 (3): 150-157

Abstract

Defibrillation plays a crucial role in the resuscitation of patients from acute life-threatening cardiac dysrhythmias causing cardiac arrest. Concerns over safety and function of defibrillators under pressure have so far prevented their routine use in clinical hyperbaric chambers. Increasing numbers of unstable and critically ill patients are being treated in such facilities for both diving and non-diving indications. This report reviews the literature relating to hyperbaric defibrillation and examines the indications, contraindications and therapeutic alternatives to this procedure.

Key Words

Equipment, hyperbaric facilities, treatment.

Inner ear barotrauma from scuba diving
Sheridan MF, Hetherington HH and Hull JJ.
Ear Nose Throat J 1999; 78: 181,184,186-187

Abstract

Inner ear barotrauma among scuba divers is believed to be caused by any of three conditions: a hemorrhage in the inner ear, a tear of the labyrinthine membrane or a perilymphatic fistula. These injuries may occur concurrently or separately. Hemorrhage and membrane rupture arc managed conservatively, while fistula requires surgical repair. In this report, we describe three cases of inner car barotrauma in scuba divers. We also discuss the proposed etiologies of this injury and the controversy over whether or not divers who have suffered an inner ear trauma can safely resume scuba diving. Although the older literature clearly suggests otherwise, we believe that scuba divers who completely recover from inner (or middle) car barotrauma may return to diving as long as they exercise caution and care.

Key Words

Barotrauma, case report, ENT, physiology, recreational diving, sequelae, treatment.

DIVING AND HYPERBARIC MEDICINE COURSES

SCHOOL OF PUBLIC HEALTH AND TROPICAL MEDICINE
JAMES COOK UNIVERSITY

COURSE IN DIVING AND TROPICAL MARINE MEDICINE
Tuesday 3rd to Friday 6th of October 2000

For further details contact
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James Cook University
Townsville, Queensland 4811
Telephone 07-4722-5700. Fax 07-4771-5032

ROYAL AUSTRALIAN NAVY
MEDICAL OFFICERS' COURSE IN UNDERWATER MEDICINE

November 27th to December 8th 2000

The course concentrates on diving physiology, fitness to dive, and emergency management of diving injuries. Practical involvement includes opportunity to dive with different types of equipment and a recompression chamber dive.

The course fee for 1999 was $1,330.00. The 2000 fee is expected to be about the same plus GST but is yet to be determined.

For information or to enrol contact
Officer in Charge
Submarine and Underwater Medicine Unit
HMAS PENGUIN, Middle Head Road
Mosman, New South Wales 2088
Tel: (61) 2 99600333. Fax: (61) 2 99604435
E-mail <Robyn.Walker@defence.gov.au>

DIVING MEDICAL CENTRE
Scuba diving medical examiner's courses

A course for doctors on diving medicine, sufficient to meet the Queensland Government requirements for recreational scuba diver assessment (AS4005.1), will be held by the Diving Medical Centre over the **Easter weekend 2001.**

Previous courses have been endorsed by the RACGP (QA&CE) for 3 Cat A CME Points per hour (total 69)
Phone Brisbane (07)-3376-1056 for further details

Information and application forms for courses can be obtained from

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

INTRODUCTORY COURSE IN DIVING AND HYPERBARIC MEDICINE

Department of Diving and Hyperbaric Medicine
Prince of Wales Hospital
Barker Street, Randwick NSW 2031

Monday 5th to Friday 16th of March 2001

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

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Decompression illness
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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>

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Content Concentrates on the assessment of fitness of candidates for diving. HSE-approved course.
October Monday 23/10/00 to Friday 27/10/00

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Content Discusses the diving-related, and other indications for hyperbaric therapy.
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Hyperbaric Thursday 2/11/00 to Friday 3/11/00
Cost $Aust 750.00

$Aust 1,300.00 for both courses taken back to back

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Unit 3 Casualty Paramedical Training.
Cost of three unit course $Aust 1, 250.00

July 2000			
Unit 1	3/7/00	to	7/7/00
Unit 2	10/7/00	to	14/7/00
Unit 3	17/7/00	to	21/7/00
October/November 2000			
Unit 1	9/10/00	to	13/10/00
Unit 2	16/10/00	to	20/10/00
Unit 3	23/10/00	to	27/10/00

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Cost $Aust 500.00

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Royal Adelaide Hospital, North Terrace
South Australia, 5000.

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