Diving and Hyperbaric Medicine

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Project Stickybeak 2001

Decompression sickness in breath-hold divers

Obesity and diving

Iatrogenic CAGE

The diving doctor's diary

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PURPOSES OF THE SOCIETY

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

MEMBERSHIP

Membership is open to all medical practitioners.
Associate membership is open to all those who are not medical practitioners but are interested in the aims of the Society, and/or those engaged in research in underwater medicine and related subjects.
Membership applications can be completed online at the Society's website <www.SPUMS.org.au>

Further information on the Society may be obtained by writing to:
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or go to <www.SPUMS.org.au>

The Society's financial year is January to December, the same as the Journal year.
The 2006 subscription will be Full Members A$132.00 and Associate Members A$66.00, including GST.
There will be an additional surcharge of $8.00 for journal postage for all members living outside Australia.

The Editor's offering

The three latest Project Stickbeak reports, 1999 – 2001, have documented 50 more tragic stories, mostly avoidable. The work collecting and collating these data is considerable. However, this is not the end of the story; these data deserve formal analysis. John Lippmann indicates in his letter that DAN Asia-Pacific is soon to employ a research assistant to collect diving accident and fatality data, which is good news. Nevertheless, this requires proper medical oversight and it behoves the Society's current officers to help ensure this happens. DAN reports from the USA lack the detail that Dr Walker has achieved and which is possible only through the establishment of a professional medical relationship with the coronial system and the police.

Whether decompression sickness (DCS) occurs in breath-hold divers has been debated for many years. Bob Wong concludes from the evidence available that indeed it does. I have seen only two convincing cases of DCS in breath-hold divers and both had been undertaking scuba dives during the same period. I suspect we will see more as freedivers push the limits of time and depth to even greater extremes. The deepest breath-hold dive recorded so far was to 209.6 metres' sea water by Patrick Musimu in 2005. Musimu floods his sinuses and Eustacian tubes beforehand so that pressure equalisation is not required during the descent, which sounds most unpleasant!

First introduced by Carl Edmonds and John Knight, *The diving doctor's diary* has appeared intermittently as an educational feature of this journal. Carl has been invited to be the first contributor to what we hope will become a regular feature once again. Here is a simple lesson that not all pain in diving is decompression sickness. A number of members of the Society have agreed to present occasional case-based educational items. However, any member is welcome to contribute if they have clinical cases from which a lesson of one form or another is to be learnt or which are simply interesting or unusual in their own right. Case histories, photos, X-rays, etc., are all welcome. Once again, may I remind you that this is *your* journal, so let's see more in the way of contributions from the wider membership.

Goble and Acott drew attention at the Noumea 2002 ASM to the fact that there were no data to show that standard scuba regulators performed adequately in an 'octopus' configuration. They expressed concern that the majority probably would not cope with two divers breathing from a single first stage in an emergency.[1] Their concerns are borne out in the Health and Safety Executive (UK) report of which the abstract and executive summary are published here. Octopus systems are still acceptable provided only high-performance equipment is used. Otherwise, the scuba-diving industry is going to have to undergo a wind change with the marketing of convenient, well-designed and independent alternative air sources. Some members of the Society have been advocating this for many years, and I recall Guy Williams discussing this in his (unpublished) presentation at the Palau ASM back in 1993.

Michael Bennett and his co-workers have spent many years gathering and critically reviewing data from randomised clinical and applied research studies of hyperbaric oxygen (HBO) in the world literature and pulling them all together in one place at <www.hboevidence.com>. In addition, Michael has been involved in a series of a dozen or so Cochrane reviews of HBO. All this work has now been documented in his doctoral thesis, which may be accessed electronically.[2] This has been a remarkable achievement. It is, of course, a never-ending task, and no doubt he is already embarked upon the next round of updating the Cochrane databases as new evidence comes in. In many clinical situations where HBO is recommended, the evidence still leaves much to be desired. It behoves those working in the field to constantly strive to accumulate the necessary research evidence to justify or reject this modality of care. In some areas and for various reasons, the gold standard of double-blinded, randomised controlled trials will never be achieved. It is important to remind oneself that evidence-based medicine involves weighing *all* the evidence available, and not simply ignoring anything less than Level 1 evidence, as is so often the case when justifying budget restraints.

Accompanying this issue is the registration documentation for next year's Annual Scientific Meeting. For only the second time in its 37-year history the Society is holding the ASM in New Zealand. Tutukaka in Northland is in the heart of one of the most beautiful coastlines in the world with a unique diving environment and the Poor Knights Islands on its doorstep. In our Guest Speaker, Neal Pollock, we have an outstanding young physiologist with a wealth of experience that has ranged from high-altitude physiology to diving in Antarctica. As the convenors, Simon Mitchell and I intend to outdo the previous New Zealand meeting, regarded by many as one of the best in recent times, with an outstanding scientific programme. It is time to break the trend of falling ASM attendances and give the 2007 meeting your strong support.

References

1 Goble S, Acott CJ. Regulator incidents: 52 incidents from the Diving Incident Monitoring Study. *SPUMS J*. 2004; 33: 30-3.

2 Bennett M. *The evidence basis of diving and hyperbaric medicine – a synthesis of the high level clinical evidence with meta-analysis*. <http://www.library.unsw.edu.au/~thesis/adt-NUN/public/adt-NUN20060808.155338/index.html>

Michael Davis

Front cover photo taken by Dr David Smart in Hobart's River Derwent is of the rare, endangered spotted handfish, *Brachionichyths hirsutus*.

Original article

Provisional report on diving-related fatalities in Australian waters 2001

Douglas Walker

Key words
Diving deaths, breath-hold diving, scuba, surface-supply breathing apparatus (SSBA), diving accidents, case reports, carbon monoxide

Abstract

(Walker D. Provisional report on diving-related fatalities in Australian waters 2001. *Diving and Hyperbaric Medicine*. 2006; 36: 122-38.)
During 2001, 12 deaths in association with breath-hold and snorkel use were identified in Australia from official sources. There were also 11 fatalities in association with scuba use, and three where surface-supply air was involved. Case summaries are presented with attention to the medical, equipment and diver performance factors. Significant adverse factors are identified and discussed for each of the groups, with comments on possibilities for reducing the number of fatalities that occur. As in previous years, the scenarios and avoidable factors are diverse. Of particular note, all three of the 'hookah' deaths were due to carbon monoxide poisoning.

Introduction

Project Stickybeak has identified and reported on deaths associated with diving in Australia since 1972 on an annual basis.[1–4] Here the deaths identified from official sources in 2001 are reported. Cases were identified from a range of sources (government, private, media) as described previously,[1] and copies of the investigation documents obtained. Summaries were made of the data and these form the basis of this review. The general methodology for searching and reviewing fatalities has been described recently.[4]

Breath-hold divers and snorkel users

CASE BH 01/1

During the trip out to the reef cay there was a talk on snorkel use and each passenger was given a leaflet on the subject. After they arrived, this 79-year-old man's wife took a ride in the glass-bottomed boat and he went to sit on the beach, then decided to go for a snorkel. One of the other passengers remembered that they had been told never to swim alone so she followed and watched him. At this time there were about a dozen people in the water with two safety officers watching from the shore. She saw that he seemed to be managing safely, taking a rest as he reached each float station. These were about 100 metres apart. She decided to return to the beach and he also chose this time to start his return, though some distance behind her. When she reached the shallower water she looked back and saw he was possibly panicking and making 'cycling' leg movements, but she was reassured when he resumed normal swimming. A short time later she again looked back and saw he was looking too still. She swam back, some 10–15 metres, and found he was unconscious with his snorkel submerged. She cried out for help and attempted to keep his head above the water. A crew member soon reached them, towed him to shore, and commenced CPR but he failed to respond to resuscitation.

Autopsy

The cause of death was given as drowning. Only moderate coronary artery atheroma and patchy myocardial fibrosis were noted, though the aorta had more severe atherosclerosis.

Comment

Breathing through a snorkel while swimming is not a natural manner of breathing; it is one that requires some adjustment of mindset. That this is so is witnessed on occasion by the death of a swimmer in shallow water while making their first snorkel swim. The witness of this tragedy suggested his 'cycling' was a panic attempt to get a footing on the sea bottom. His swimming and snorkel experience is unknown but it is highly likely it was minimal, if any at all. It is probable the outcome would have been critically improved had he thought to remove his snorkel and breath 'naturally'.

Summary

SNORKELLING; EXPERIENCE UNKNOWN; POSSIBLE PANIC; INHALATION OF WATER; DROWNING; SOME CORONARY ATHEROMA; NO HEALTH HISTORY; CARDIAC FACTOR?

CASE BH 01/2

This 61-year-old woman was among 41 passengers making a day trip to a reef cay. There was the regular health questionnaire and safety talk for those intending to snorkel, and they were advised to wear a buoyancy vest unless very experienced. Neither she nor her partner reported any adverse health history. They were offered and accepted the suggestion of wearing wetsuits. It is not recorded whether she had ever previously used a snorkel.

After they disembarked at the cay they snorkelled for a time then took a trip in the glass-bottomed boat to view the coral. After lunch back on the boat there was an announcement that those who were not confident could come on a guided snorkel trip to the outer reef of the cay. The skipper was watching their water entry and was reportedly impatient at the victim's slowness in entering the water to join the others after her husband had tightened her buoyancy vest. Once in the water she found the vest was riding up over her head, having been incorrectly fitted. She returned to the boat's stern board but was unable to correct the problem and the skipper helped her remove the vest. She said she was anxious without it and got him to promise to remain close to her. However, he then boarded the outboard inflatable tender and drove away to collect all the others of the snorkel group around him. She swam alone for about 10–15 minutes then saw the glass-bottomed boat and called out that she had swallowed some water and wanted to be taken aboard.

The crewman on this boat offered no assistance and it was the passengers who dragged her part into the boat when she was unable to pull herself aboard. They became anxious when she started making gurgling noises and said she couldn't breathe, and they became vocally aggressive when the crewman refused to take any action. Ultimately their reaction forced him to inform the skipper, who drove over to them and helped pull her fully into the boat. Their resuscitation efforts were not helped by their inability to open the oxygen bottle because of the absence of a spanner. Despite the vociferous objections of the passengers the boat was then driven back to the main vessel bypassing another vessel that might have provided assistance. No call for emergency assistance was made because the two-way radio did not work.

Autopsy

The cause of death was reported as salt-water drowning. She was described as obese and sturdy.

Comment

This tragedy was an example of what can happen if there is a gross failure to act professionally by those responsible for the safety of others. This case is particularly unusual in that there were apparently serious inadequacies by more than one member of the crew in the level of supervision and assistance given to a passenger who requested, and indeed was promised, specific attention. The company concerned was subsequently prosecuted, but this did not help the deceased. It is the obvious responsibility of those with a duty of care concerning the safety of passengers to deliver such care as is necessary. Fortunately the correct level of supervision and assistance is almost always provided.

Summary

SNORKELLING; BUOYANCY VEST REMOVED; LACK OF SUPERVISION BY SKIPPER; SEPARATION; LACK OF ASSISTANCE BY CREWMAN; NO SPANNER FOR OXYGEN CYLINDER; INOPERATIVE RADIO; CREWMAN REFUSED TO SEEK ASSISTANCE FROM OTHER VESSEL; DROWNING.

CASE BH 01/3

This flooded, abandoned, old open-cast mine was a favourite swimming area for the local community. It varied in depth from shallow to about 10–15 metres, where there was a collection of dumped mine equipment. A faded notice warning people to keep out had for long been ignored. The 25-year-old victim was reported to have been drinking heavily the previous evening and had a couple of cans of beer that morning before joining his friends at the mine, plus another can after arriving. He was described as being a professional diver, probably breath-hold for *beche de mer*. There was a buoy marking the position of the dumped equipment.

The length of his first dive was alarming his friends until he surfaced after about two minutes. Although his back was to observers, he is thought to have removed his mask, washed it out, replaced it and then submerged again. They believed he was swimming underwater to a shallow ledge, but he failed to reappear. After four to five minutes a search was started, but they had only one mask and it tended to leak. The police were informed but postponed a search till police divers could attend the next day. This stimulated the locals to organise a search to be made using borrowed hookah equipment. This was successful on the second search dive. The body, mask in position, was lying on the debris below.

Autopsy

The cause of death was reported as drowning following a post-hyperventilation blackout. His blood alcohol level was 222 mg.100ml.

Comment

It is known from swimming-pool deaths that swimming activity can continue after consciousness is lost and this could explain the sequence of events described. Among those who find they can extend their underwater time during breath-hold dives there are always some who refuse to believe that pre-descent hyperventilation has a potential

Table 1. Summary of diving-related fatalities
(BH – breath-hold, BNS – buddy not separated, BSB – buddy separation before incident,

Case	Age	Training	Experience	Dive group	Dive purpose	Depth (metres) Dive	Depth (metres) Incident
BH 01/1	79	Not stated	Not stated	BSB	Recreation	Not stated	Surface
BH 01/2	61	Not stated	Not stated	GSB	Recreation	Not stated	Surface
BH 01/3	25	Nil	Experienced	Solo	Recreation	15	Ascent
BH 01/4	76	Nil	Nil	GSB	Recreation	Not stated	Surface
BH 01/5	21	Nil	Nil	BSB	Spear fishing	Not stated	Not stated
BH 01/6	44	Not stated	Experienced	Solo	Spear fishing	3	3
BH 01/7	35	Nil	Nil	BSD	Recreation	Not stated	Surface
BH 01/8	43	Nil	Some	BSB	Spear fishing	5	Surface
BH 01/9	81	Not stated	Not stated	BNS	Recreation	5	Surface
BH 01/10	51	Not stated	Experienced	Solo	Spear fishing	Not stated	Surface
BH 01/11	38	Not stated	Not stated	GSB	Food gathering	Not stated	Surface
BH 01/12	32	Not stated	Experienced	BSB	Spear fishing	25	Ascent

cost – death. Clearly he should not have been freediving following heavy drinking.

Summary

SNORKELLING; BREATH-HOLD DIVER; EXPERIENCED; POST-HYPERVENTILATION BLACKOUT; ALCOHOL; DROWNING.

CASE BH 01/4

During this 76-year-old man's visit from overseas to a childhood friend they decided to include a live-aboard trip to view the Barrier Reef. During this trip they were offered the opportunity to snorkel over the reef and given a safety briefing. The 12 who chose to take up this offer were taken to the selected area in a tender, given further advice, and had their names listed on a slate board before entering the water. The tender remained in the centre of the swimming area, which contained an inflated tyre as a rest station and had a safety watcher making frequent head counts. There were also watchers on a second tender.

After about 10 minutes it was noticed that one swimmer had not moved since the previous check and an immediate investigation found the victim floating face down, head submerged, and unconscious. There was no warning disturbance, the person "had just gone limp". Resuscitation efforts were not successful.

Autopsy

The heart was greatly enlarged and there was severe coronary atherosclerosis. The diagnosis was of myocardial infarction but the autopsy report is limited to these data.

Comment

The victim's friend later recalled that he had made frequent rest stops during their earlier sightseeing, though had not made any complaints of health symptoms. It was established that he was taking (un-named) medications for his heart, had his hypertension checked monthly, and had achieved normal blood pressure at the last check. This health problem may have been the reason why he had airfare insurance but no travel health insurance. There is no documentation concerning whether he was asked to fill in a health questionnaire when booking this trip, but even had he disclosed his health condition it is uncertain whether he could have reasonably been refused the opportunity to snorkel. The safety arrangements of this live-aboard travel organisation appeared to be excellent.

in Australian waters in 2001, breath-hold incidents
BSD – buddy separation during incident, GSB – group separation before incident)

Weight belt On	Kg	Bouyancy vest	Remaining air	Equipment Tested	Whose	Comments
None	n/a	Nil	n/a	n/a		Drowning. Possible cardiac death.
None	n/a	Off	n/a	n/a		Drowning (delayed). Possible cardiac arrhythmia.
None	n/a	Nil	n/a	n/a		Post-hyperventilation blackout.
None	n/a	Nil	n/a	n/a		Cardiac death.
None	n/a	Nil	n/a	n/a		Drowned. Inexperienced. Rip area. Solo.
On	5	Nil	n/a	n/a		Severe head trauma.
None	n/a	Nil	n/a	n/a		Cardiac death.
On	7	Nil	n/a	n/a		Drowned.
None	n/a	Nil	n/a	n/a		Cardiac death.
Off	4	Nil	n/a	n/a		Cardiac death.
None	n/a	Nil	n/a	n/a		Drowned. Borrowed equipment.
On	Not stated	Nil	n/a	n/a		Post-hyperventilation blackout.

Summary

SNORKELLING; SILENT DEATH; WELL-SUPERVISED AREA; UNREVEALED HEALTH HISTORY; SEVERE CARDIAC DISEASE; HYPERTENSION ON MEDICATION; MYOCARDIAL INFARCTION.

CASE BH 01/05

When this 21-year-old man arrived at the beach several of his friends were already there and swimming either in the sea or in a rock pool. They had one set of mask, snorkel and fins and he borrowed this equipment in order to go spear fishing. His friends thought he was inexperienced in this sport. After watching him swimming around for a time, they went for a walk along the beach. When they failed to see him on their return they became alarmed and informed the lifesavers. When he had entered the water there had been two others spear fishing nearby, but they had soon left the water. It was two hours before his body was found floating in shallow water.

Autopsy

No coronary or other disease was identified. Death was recorded as drowning.

Comment

A lifesaver noted that there was a permanent rip in the area of the rocks where the victim had been swimming, and a police officer suggested he might have drowned when he became trapped under a rock ledge, but his inexperience was the most likely critical factor, presumably allied to the water conditions.

Summary

SNORKELLING; INEXPERIENCED; SPEAR FISHING; SOLO; RIP AREA; DROWNED.

CASE BH 01/6

Although it was his usual practice to have a float and ‘diver down’ flag, on this occasion this 44-year-old spear fisherman had omitted to use them. He had no success when diving off the beach so had moved to a rocky area close to the entrance to a small harbour that was frequented by recreational fishermen. They used aluminium boats with powerful outboard motors and on this day two decided to return to harbour at about the same time. The local council had put up signs warning boats about the presence of swimmers in the harbour approach waters but these had been repeatedly vandalised and destroyed.

The first intimation of the tragedy was when the spear fisherman's wife became alarmed at his failure to return home. The police were informed and a police boat took police divers to the rocky area where a witness had earlier seen a spear fisherman. They anchored their boat in three metres of water over a rocky ledge at the edge of this area and the victim's body was found on the sea bed close by at a depth of about four metres' sea water (msw). When found, his weight belt was on, his facemask lacked its faceplate and his snorkel had lost its mouthpiece. His gun was damaged and he had signs of trauma on the front of his skull. It was clear this was likely to be from a propeller blade. Investigation located a workshop with a propeller for repair that had been damaged the day of the fatality.

Autopsy

There was a head laceration involving the frontal and left parietal regions approximately 17 cm long with several protruding fragments of the skull. Brain tissue had escaped through this injury. There was a second 5 cm wound over the occipital scalp that did not penetrate the outer table of the skull. There were abrasions on his forehead, a cut in the left thenar eminence, a superficial linear defect over the dorsum of the left forearm, and several other lacerations and abrasions to the upper limbs. The heart appeared healthy with some patchy eccentric atheroma with up to 75% luminal narrowing of the proximal left anterior descending artery and up to 50% of the right coronary artery. The left kidney was congenitally absent. There was pulmonary congestion and blood-stained fluid in both pleural cavities. No rib fractures or pneumothoraces were identified. This finding led the pathologist to suggest cardiac failure preceded the fatal brain injury, though he suggested some delay could well occur before he died from such trauma.

Comment

The death of this spear fisherman from trauma was a consequence of errors by both the victim and the driver of the motor boat. The two boat drivers denied they had been racing each other. The boat's owner was reluctant to admit either that there was a high probability he had hit the diver or that he had been too close to this rocky area. It would be difficult to see a diver's black wetsuit from a speeding boat, but the course taken was incorrectly close to the rock outcrop area. The boat's 'skeg', which projects below the propeller, was broken leaving the propeller exposed. The significance of this broken skeg is unclear, but had it been present the injury caused to the victim might have been less severe. It was natural that the evidence as to whether the driver of the boat had been racing his friend back to the ramp would be disputed, as would the question concerning what a careful boat driver would have seen of a snorkeller at the surface, taking into account the surface wavelets and position of the sun. The evidence of a RAN officer was that identification of such a situation as in this fatality would be easy for anyone keeping the correct lookout; an opinion disputed as unrealistic. The failure on the part of the diver to have a float and 'diver down' flag was an unfortunate break from his usual practice and placed him in danger. The conjecture by the pathologist regarding cardiac failure provided fertile grounds for legal questioning at the inquest.

Summary

SNORKELLING; SPEAR FISHING NEAR HARBOUR ENTRANCE; SOLO; NO FLOAT OR DIVE FLAG; HIT BY BOAT PROPELLER; POSSIBLY BOAT RACING AND INADEQUATE LOOKOUT; FATAL SEVERE HEAD INJURY.

CASE BH 01/7

A friend had suggested this 35-year-old man come to watch while he dived to collect abalone and crayfish. As he was about to get into his car afterwards he asked the victim if he would like to dive with him, an offer he immediately accepted. Nothing is known concerning his swimming ability or whether he had previously used a snorkel, but it is believed this may have been his first use. After he had donned the offered wetsuit and mask, snorkel and fins, they prepared to enter the water. However, an unexpected wave washed them both off the rocks and they then swam about 20 metres out to sea. The buddy started diving and soon collected 20 abalone and a crayfish, then noticed that the victim was treading water in an attempt to keep his head above the surface. He managed to reassure him and then dived again. When he surfaced he saw the victim floating face down and unconscious, the snorkel firmly clenched in his jaw and its end under the water.

He attempted to tow his friend back to the rocks and get him ashore but the current and the waves over the rocks defeated his efforts and it was a diver dropped from a rescue helicopter who eventually recovered the body.

Autopsy

The autopsy showed that he had had triple bypass surgery and that the left anterior descending coronary artery showed 80% narrowing. There was evidence of an acute myocardial infarction indicating that this was not a simple drowning. It was also found that he had suffered a previous myocardial infarction.

Comment

This case is particularly unusual in relation to the health history at his age, 35 years. Whether he was having any symptoms from his cardiac condition or whether the friend was aware of his health problems is not recorded. Even had he known of the victim's medical history, he could not be blamed for the deceased accepting the offer of the use of the equipment to swim with him. While it is easy with hindsight to question his failure to recognise the danger of leaving anyone who was having difficulty keeping his head above the water, he evidently believed the situation had stabilised before he dived again.

Summary

SNORKELLING; INEXPERIENCED; SOLO WITH SCUBA DIVER BELOW; PREVIOUS AND RECENT MYOCARDIAL INFARCTS AND HISTORY OF TRIPLE BYPASS OPERATION; SUDDEN CARDIAC DEATH.

CASE BH 01/8

This 43-year-old man had done some scuba diving, but was untrained, and some breath-hold spear fishing, but no details are available concerning his experience beyond the statement that he had accompanied his recent friend on seven occasions. This was the first time he had worn his new weight belt. The water was calm close to the waterway wall where they made their water entry and they kept a distance apart to avoid entangling their float lines. When he became swept away from the calm area into the middle of the channel he called for help. His friend had previously advised him that if this occurred he should try to get close to the rocks as the current was weaker there, but he evidently recognised he was unable to do this.

There were two boats anchored in the waterway about 15 metres from him and one of the men fishing from them dived into the water to assist him, but he sank from sight before he could be reached. A search was made and he was found lying on the sea bed, all equipment in position, holding his spear gun.

Autopsy

The cause of death was given as drowning, with no adverse health factors identified.

Comment

The view of his buddy is probably correct: that the critical factors were excessive weight on his belt and his failure to ditch it. Without this weight he could have swum towards the wall, where there was less current, and survived. Probably he panicked when unable to swim against the strong current to regain the calmer water. His inexperience would explain a panicked rather than a reasoned response to the situation.

Summary

SNORKELLING; SPEAR FISHING; PROBABLY INEXPERIENCED; CAUGHT IN CURRENT; FAILED TO DITCH WEIGHT BELT OR SPEAR GUN; NO HEALTH FACTORS; DROWNING.

CASE BH 01/9

This 81-year-old man and his wife took a boat trip to view the Great Barrier Reef. He regretted his failure to snorkel over the coral due to a reluctance to do so from a boat. However, after their return to the resort island, he decided he would go for a snorkel swim near its jetty. There was a notice here warning against swimming, no doubt because of tourist boats, but locals told him it would be safe. His wife entered the water first as he had limited confidence in entering alone. When she looked back to check on his progress she was alarmed to see him under the jetty, holding onto one of its pylons, gasping for breath. He appeared to be disoriented. She managed to pull him from under the jetty and called for help but he rapidly started talking gibberish, then died. He had expended minimal effort in the short distance he had swum.

Autopsy

There was only mild to moderate coronary atheroma and mild myocardial interstitial fibrosis, so the diagnosis was made of acute cardiac failure due to ventricular fibrillation.

Comment

Before a holiday to New Zealand not long before, he had a check from his cardiologist, whom he attended for an irregular heart rhythm. He was assured that he had no heart damage and to continue his diltiazem. However, whilst on holiday, he suffered an episode of heart symptoms and was fully investigated. He was reassured that this was probably due to a trip in an unpressurised plane. As two cardiologists appear to have down-played the significance of his arrhythmia his decision to swim using a snorkel was legitimate and the outcome unpredictable.

Summary

SNORKELLING; COLLAPSE SOON AFTER WATER ENTRY; HISTORY OF CARDIAC ARRHYTHMIA ON MEDICATION; RECENT CARDIOLOGY TESTS; SUDDEN CARDIAC DEATH.

CASE BH 01/10

The discovery of this 51-year-old man's body was by chance, two tourists having stopped to admire the view and then seeing his body on the beach, head in the water. It was noted that his wetsuit was unzipped and part removed, his weight belt and other equipment off and lying on the beach next to him. He was known to be an experienced and careful spear fisherman so the fact that his spear gun and crayfish gun were both loaded when found was taken as evidence that this had not been a routine return to the beach. It was suggested that he had probably felt unwell, the likely reason for him to leave the water. It was discovered that he had reported experiencing three episodes of chest pain in the past two weeks and had been reassured they were not cardiac in origin after several (unspecified) tests.

Autopsy

This revealed the presence of an enlarged heart and 90% narrowing of an area of the left anterior descending coronary artery. There was also a small scar in the wall of the left ventricle suggestive of a past myocardial infarction. His death was regarded as due to a further infarction.

Comment

The evaluation of the significance of symptoms presented by a patient is undoubtedly influenced by many factors, and in this instance appears to have been incorrect. It is possible the outcome would have been the same even had he not been alone at the critical time, but being alone reduced his chance of acute medical care. Possibly more significance should have been placed on chest-pain symptoms in a man of this age.

Summary

SNORKELLING; EXPERIENCED SPEAR FISHERMAN; SOLO; EXITED WATER AND MANAGED TO PART REMOVE EQUIPMENT BEFORE DIED; CHEST PAINS PAST TWO WEEKS; CORONARY ATHEROMA; ACUTE MYOCARDIAL INFARCTION; SUDDEN CARDIAC DEATH.

CASE BH 01/11

This apparently healthy 38-year-old man was with a family group that was hunting for kina off a rocky shore. The conditions were not good, the water rough and murky, indeed the police later said conditions were unsuitable for this activity. Nothing is recorded concerning his experience with snorkel or his general swimming ability. One of the group, reportedly a good swimmer, was knocked over by the waves several times while standing on a rock in the water. It is unclear from the available information whether he was finding the kina by ducking his head underwater or truly breath-hold diving for them. The former practice is thought the more likely.

They had brought a dog to the beach with them and it was noticed to be leaving the beach in pursuit of another dog. When the victim failed to make this clear to the dog's owner he decided to swim back to the beach to collect it, handing his spear to a female family member snorkelling near him. None of the others appeared to have noticed his absence until the alarm was raised by witnesses on a hillside overlooking the beach. They saw a body floating face down and both raised the alarm and raced to give assistance, joined by some spear fishermen who were nearby. He failed to respond to their resuscitation efforts.

Autopsy

The cause of death was given as drowning. He was found to have mild ventricular hypertrophy, the significance of which was unknown.

Comment

Neither his swimming ability nor his previous experience using a snorkel are known, but it is apparent that members of the group were overconfident of managing themselves in the rough water.

Summary

SNORKELLING; SEPARATION; EXPERIENCE UNKNOWN; ROUGH WATER; MILD VENTRICULAR HYPERTROPHY; DROWNING.

CASE BH 01/12

This 32-year-old man and his friend were both experienced spear fishermen but he was by far the better, with a usual pattern of diving to a depth of 20 msw. Their half-cabin boat was anchored off a small island, and while the friend remained in the shallower water near the island the victim hunted in the deeper water. His friend remained unconcerned at their separation until, about 60 minutes after their last sighting of each other, a passing dive boat noticed his float in the rough water close to the island. He realised that his friend would never let go of his spear gun and float so something must have happened to him. A search was undertaken and his body was found on the sea bed, weight belt and mask in place.

Autopsy

The cause of death was drowning, undoubtedly following a hyperventilation blackout.

Comment

It is breath-hold divers of superior ability who are most likely to put themselves at risk of post-hyperventilation anoxic loss of consciousness. Spear fishing does not lend itself readily to realistic buddy diving so scenarios such as this one are not unexpected from time to time. Many freedivers do not follow the 'one up, one down' rule for buddying whilst spear fishing.

Summary

SNORKELLING; EXPERIENCED SPEAR FISHERMAN; SEPARATION/SOLO; POST-HYPERVENTILATION BLACKOUT; DROWNING.

Scuba divers

CASE SC 01/1

This 29-year-old man had made only two dives since his course two or three months previously. His buddy had been diving 'occasionally' for 17 years, and though he had never been trained in scuba diving he claimed experience in using breathing apparatus because of being a fireman. The scuba equipment the victim was to use was borrowed from his buddy. Their plan was to catch crayfish and after entering the water off a rock ledge they swam out about 30 metres using their snorkels before changing to scuba. The depth was about 10 msw. They had been swimming vigorously in their ineffective pursuit of the crayfish. After a time, he showed his contents gauge to his buddy as it showed a low air situation, and they started to ascend. When the buddy reached the surface he was surprised to find his friend already there as he had been lectured by him before the dive on the need to ascend slowly, a matter evidently well stressed during the recent course.

He told his buddy he was feeling unwell so they inflated their BCDs and started to snorkel back to the shore. After they had swum about three metres he again said he felt unwell, then grabbed hold of his buddy's equipment and did not reply when spoken to. During the struggle that followed the buddy's tank slipped from its harness so he was unable to reach his regulator and had to continue on snorkel. He was seriously hampered by the weight of his friend, who was now unconscious, and called for help. They were being repeatedly submerged by the swell so he decided to detach his friend, return to shore to remove his equipment, then return unencumbered. When he returned he found a swimmer trying to keep his friend's head above the water. Together they brought him to a rock ledge where others helped bring him ashore. Their resuscitation efforts were hampered by his regurgitation and were unsuccessful. A witness described the water as being rough and cold.

Autopsy

Pre-autopsy X-rays showed widely disseminated gas bubbles within the brainstem and meningeal vessels, major arteries, the pulmonary arteries, and within both cardiac ventricles. Death was due to cerebral arterial gas embolism (CAGE).

Comment

He was inexperienced, and initiated his ascent when low on air. It would appear that he ascended rapidly, as he was already on the surface when his buddy surfaced. The fatal embolism is likely to have occurred at this time from lung barotrauma sustained during ascent. A valiant attempt at rescue and resuscitation was made by his buddy and others. The equipment was checked and no significant faults were found. There was a delay of several minutes following his reaching the surface before he lost consciousness.

Summary

SCUBA; NEWLY TRAINED AND INEXPERIENCED; BORROWED EQUIPMENT; RAPID LOW-AIR ASCENT; CONSCIOUS AT SURFACE INITIALLY; INFLATED BCD; GRABBED BUDDY CAUSING DANGER TO HIM; LOSS OF CONSCIOUSNESS THEN SEPARATION; BUDDY'S TANK SLIPPED FROM HARNESS; DELAYED RESCUE; CAGE.

CASE SC 01/2

The 29-year-old male victim had trained two years previously but nothing is recorded concerning his subsequent diving experience. On this day he was with his brother, he to scuba dive and his brother to snorkel above and act as a 'one-remove' buddy. Their water entry was off rocks and he then descended into a kelp-filled gulley about two metres deep. He soon became caught in the kelp, which entangled both him and his regulator and tank. His brother saw what had occurred but in his anxious haste he dropped his knife, and the victim had not brought one. There was some delay before his cries for help were understood by others and when help arrived it was too late. The victim was found head-down, his fins at the surface and regulator out of his mouth. He was cut free but was beyond resuscitation, having drowned.

Autopsy

The cause of death was given as drowning. The coronary arteries were healthy. Although there was a history of asthma there was nothing to suggest this or 'flu-like' symptoms over the previous two days contributed to his death.

Comment

The tragic outcome of this dive could so easily have been avoided. Kelp should always be entered with caution, as entanglement is always possible, though proper dive technique should avoid this. In this incident, the buddy's panic response led to him dropping the knife that may have offered the only chance of getting the victim free.

Summary

SCUBA; TRAINED BUT UNKNOWN EXPERIENCE; ENTANGLED IN KELP NEAR SURFACE; 'BUDDY' PANICKED AND LOST KNIFE; TWO DAYS OF 'FLU-LIKE' SYMPTOMS AND ASTHMA HISTORY NOT FACTORS; DROWNED.

CASE SC 01/3

Although claiming 25 years' scuba diving experience, this 42-year-old's actual experience is not recorded. However, she had taken a 'refresher course' three months prior to this dive. She was among 14 divers on a commercially run boat dive. The divemaster, an instructor, was her buddy and he

Table 2. Summary of diving-related fatalities
(BNS – buddy not separated, BSB – buddy separation before incident, BSD – buddy separation during incident,

Case	Age	Training	Experience	Dive group	Dive purpose	Depth (metres) Dive	Depth (metres) Incident	Weight belt On	Weight belt Kg
SC 01/1	29	Trained	Nil	BSD	Cray fishing	10	10	On	Not stated
SC 01/2	29	Trained	Not stated	Solo	Recreation	2	2	On	Not stated
SC 01/3	42	Trained	Experienced	BSB	Recreation	20	20	On	Not stated
SC 01/4	58	Trained	Nil	BNS	Cray fishing	9	Ascent	On	14
SC 01/5	64	Trained	Experienced	BSD	Recreation	11	1	On	6
SC 01/6	30	Nil	Not stated	BNS	Recreation	1.5	Surface	Buddy off	Not stated
SC 01/7	38	Trained	Some	GSB	Recreation	20.5	Surface	On	2
SC 01/8	26	Nil	Nil	BNS	Class	9	9	On	9
SC 01/9	56	Trained	Some	BSB	Recreation	10	Ascent	On	8
SC 01/10	65	Trained	Experienced	BSB	Recreation	6	Land	Off	Not stated
SC 01/11	55	Trained	Some	BNS	Recreation	Not stated	Surface	On	Not stated
H 01/1	23	Some	Experienced	Solo	Work	5	5	On	Not stated
H 01/2	35	Nil	Nil	BNS	Cray fishing	Not stated	Not stated	On	Not stated
H 01/3	32	Nil	Nil	BNS	Cray fishing	Not stated	Not stated	On	Not stated

noted she seemed a little stressed after her water entry but soon settled. She had a problem with water entering her mask but this was resolved by her buddy who removed the edge of her hood from under the mask. They exchanged frequent 'OK?' signals and experienced no further problems.

At a depth of 20 msw her gauge showed only 70 bar air remaining. Her buddy had plenty, so it was agreed that she would ascend alone while he continued hunting for crayfish. He watched the first five to six metres of her ascent and, satisfied at her progress, stopped watching. When he surfaced 10–15 minutes later he was surprised to be asked where his buddy was. He was told, in answer to his questions, that a call for help had been heard near some rocks 10–30 metres from the boat before he surfaced. He removed his equipment and left it on the boat, then snorkelled to the rocks and began a search. He soon saw her lying on the sea bed five metres below but was unable to dive to her because he was no longer wearing his weight belt. Another diver recovered her body but resuscitation efforts were without success. Her weight belt was in position, BCD not inflated. After her body was recovered there was just sufficient air to inflate her BCD but there was reportedly insufficient to inflate it five metres underwater. Subsequent inquiries revealed a history of a spontaneous pneumothorax in 1996. She was reported to be a 'mild asthmatic' and to be suffering from some abdominal pain following a recent gynaecological operation. She had a 'dive medical' before the refresher course but it is not known whether she revealed her medical history as the doctor, not on the SPUMS list, had kept no records.

Autopsy

Autopsy did not reveal any evidence of CAGE, though there were some adhesions in the right pleural cavity. The cause of death was given as drowning.

in Australian waters in 2001, scuba and hookah incidents
CAGE – cerebral arterial gas embolism, GSB – group separation before incident, H – hookah, SC – scuba)

Bouyancy vest	Remaining air	Equipment Tested	Equipment Whose	Comments
Inflated	Low	No fault	Own	CAGE.
Not inflated	Not stated	Not stated	Own	Drowned. Entangled in kelp. Buddy dropped only knife.
Not inflated	Low	NAD	Own	CAGE? Spontaneous pneumothorax. 1996 asthma. Post-operation abdominal pains.
Partially inflated	Low	Not tested	Borrowed	CAGE. Obese, BMI 32. History of chest injury and mild asthma.
Inflated	Nil	No fault	Own	Drowned. Separation. Fatigue. Tight wetsuit. Infected knee.
Buddy inflated	++	Not tested	Borrowed	Drowned. Nil training/experience. Surface rough. BCD not inflated, weight belt on. Could have stood up.
Inflated then deflated	Nil	No fault	Hired	Drowned. No diving for 4 to 5 years. Recent back pain. Deflated BCD.
Buddy inflated	++	Fault	Borrowed	CAGE. Under intruction. Damaged mouthpieces. Medical history.
Inflated	+	NAD	Hired	CAGE.
Not inflated	Low	NAD	Own	Cardiac death.
Buddy inflated	++	NAD	Own	Cardiac death.
Nil	n/a	Fault +	Employer	CO poisoning.
Nil	n/a	Fault +	Borrowed	CO poisoning.
Nil	n/a	Fault +	Borrowed	CO poisoning.

Comment

That her buddy, an instructor, agreed to their separation and her solo ascent may not appear anything unexpected or out of the ordinary but it was a breach of basic diving safety rules. Although she was not technically in a 'low air' state she appears to have been out of air at the surface. The failure of those in the dive boat to react to a call for help is unacceptable behaviour. Despite the lack of autopsy evidence, judging from the description of events it is possible she had already suffered a cerebral arterial embolism at that time. Whether she died from this or was incapacitated and then drowned cannot be decided, and neither can the significance of the history of asthma, pain from a recent operation, or surgical treatment for a spontaneous pneumothorax. The failure of the doctor to keep records is noted.

Summary

SCUBA; RECENT REFRESHER COURSE; SEPARATION FOR SOLO ASCENT WHEN LOW AIR; BUDDY AN INSTRUCTOR; NO RESPONSE FROM BOAT TO CRY FOR HELP; WEIGHT BELT ON; MINIMAL REMAINING AIR; POST-OPERATION ABDOMINAL PAIN; HISTORY OF SPONTANEOUS PNEUMOTHORAX; POSSIBLY CAGE; DROWNING.

CASE SC 01/4

This 58-year-old man had not dived often since his course some years previously. A friend loaned him a 90 cu ft tank and other equipment for him to come on a dive to catch crayfish. There were seven divers in the boat and he was in a trio team. After about 20 minutes at 9 msw they saw a crayfish in a crevice and allowed him to try to snare it, but he was unsuccessful. When they checked their contents gauges his read 50 bar, theirs 100 bar, so they arranged for one to remain on the sea bed while the other accompanied him as he ascended.

The buddy kept a close hold on him during the ascent to prevent it being too rapid, though he was not ascending fast. At the surface he saw that he had half inflated his BCD and told him to use his regulator as he lay on his back and finned to the boat. He then descended to rejoin the third member of their group. When they surfaced soon after, they saw him floating face up near the mermaid line and it was only after removing their equipment in the dive boat that they realised he was not just lying there enjoying the sunshine. When reached, they found his BCD was tensely inflated and he was unconscious. They had difficulty pulling him into the boat because he was a large man.

Autopsy

X-rays taken before the autopsy showed a fluid level in the right atrium. Some air escaped when the heart was opened under water. There was a small fibrous pleural adhesion in the left pleural cavity, thought to result from a previous road accident. His heart was enlarged, the left ventricle wall thickened, and he was described as being obese (BMI 32). Cause of death was CAGE.

Comment

The buddy acted correctly, ascending with him, but should not have separated from him on the surface. Despite the apparently correct ascent and absence of any indication of distress at the surface, a CAGE occurred. He possibly experienced some intimation of this and fully inflated his BCD. The constricting effect of a hyper-inflated BCD may have been a critical adverse factor. The controlled nature of his ascent would not have prevented pulmonary barotrauma if he failed to exhale correctly during the ascent. There is nothing to clearly implicate his medical history of asthma or the small adhesion in his death. The police did not test the equipment. There was a short delay between his surfacing and the onset of the CAGE symptoms.

Summary

SCUBA; TRAINED; INEXPERIENCED; CONTROLLED LOW-AIR ASCENT ACCOMPANIED BY BUDDY; SEPARATION AT SURFACE WITH HALF-INFLATED BCD NEAR BOAT; FOUND FLOATING ON BACK WITH OVER-INFLATED BCD; OBESE; HISTORY PAST RTA CHEST INJURY AND ASTHMA; CAGE.

CASE SC 01/5

This 64-year-old woman and her husband had been making occasional dives for about 10 years, a total of about 100 dives. She was reportedly in good health, but had undergone left knee surgery six weeks earlier. When they booked for a live-aboard dive trip they were assessed before being accepted. After the second day's diving she said she was feeling more fatigued than she had expected. The next day the couple made a successful reef dive and after surfacing they decided to swim back to the boat just under the surface, the husband a little in advance of his wife. He began to feel breathless and found he was low on air, indeed had too little to inflate his BCD so he orally inflated it. Then he noted the absence of his wife and signalled to the dive tender. A short search located her, weights on, floating a little below the surface. It is not stated whether her BCD was inflated.

Autopsy

Her heart and coronary arteries were healthy, the cause of death being given as drowning.

Comment

It is probable that the combination of fatigue from swimming against the current, a tight wetsuit, low-air status such that, like her husband, she could not fully inflate her BCD, and failing to think to drop her weight belt, led to her drowning. She may have been distracted by pain from an infection in the left knee mentioned by her husband, but there was no complaint of any problem while diving and the condition of the knee was not reported at the autopsy.

Summary

SCUBA; TRAINED; MODERATE EXPERIENCE; LOW-AIR SWIM JUST BELOW SURFACE; SEPARATION; TIGHT WETSUIT; PAINFUL LEFT KNEE DUE TO POST-OPERATIVE INFECTION; FAILED TO DROP WEIGHT BELT; SILENT DEATH; DROWNING.

CASE SC 01/6

This 30-year-old man met a friend at the beach and after they had been surfing together he suggested they go for a scuba dive. The friend had been diving for many years, mainly for golf balls, but only taken a course a year ago. He knew that the victim had a wetsuit and surfed and never thought to ask whether he was trained, or had ever used scuba previously.

Their plan was for this to be a snorkel dive but wearing scuba 'for use in case of emergency'. The buddy loaned the victim the scuba equipment and checked that his equipment was in order before they entered the water from a beach. They waded out about 10 metres before reaching sufficiently deep water to begin snorkelling. There was a slight swell at the beginning of the surf zone. They were only two to three metres apart at this time. A short time later the buddy heard a woman shouting out that someone was in trouble. He saw her returning to the beach and two swimmers, about 40 metres from the beach, trying to support another person's head above the water. There were 0.5–1 metre high waves breaking over them.

When the buddy reached the scene he heard his friend saying "I'm freaking out, I'm going in". The buddy jammed the regulator into the victim's mouth but he spat it out. It was possible here to stand on the seabed with head above the surface. They started to return to the beach, the victim 'dog paddling', but they became separated. The victim was found underwater after a short search, and the buddy released his weight belt and inflated his BCD to bring him to the surface, then brought him ashore with assistance from others.

Autopsy

There was an area of 50% narrowing of the left anterior descending coronary artery of unknown significance. The cause of death was given as drowning.

Comment

Panic, possibly due to total ignorance of scuba use, occurring in the rough but relatively shallow water, prevented him from remembering the life-saving response of ditching his weight belt and inflating his BCD and/or going onto scuba. As the victim's buddy had dived for years before obtaining training, not checking the victim's experience beforehand was understandable, though unwise. Whether his coronary condition affected his response cannot be known. This was really a snorkelling death whilst wearing scuba.

Summary

SCUBA BUT SNORKELLING; UNTRAINED; FIRST USE SCUBA; BORROWED EQUIPMENT; SEPARATION THEN PANIC IN ROUGH WATER; FAILED TO DROP WEIGHT BELT OR INFLATE BCD; FAILED TO GO ON SCUBA; COULD HAVE STOOD UP; SOME CORONARY ATHEROMA; DROWNING.

CASE SC 01/7

This 38-year-old diver had not dived for over four years because of a back injury. There was apparently no significant disability now and he was accepted for this live-aboard dive trip after disclosing his medical history. The instructor on the boat accepted his evidence of 40 previous dives as reason not to formally test his ability but a fellow passenger, his assigned buddy for the first three dives, was a qualified instructor and was satisfied with his ability. The passengers were transferred from the first boat following these dives to the one with sleeping quarters, and the first night he joined the guided night dive and was assessed as a confident diver. The next morning he was in a trio group, one member the boat's instructor, which experienced a strong current, and after they returned to the boat he took a rest.

That afternoon the same trio group dived again and the current was still strong. During this dive the instructor member became separated but they met again after surfacing. They signalled for the tender to collect them as they floated with inflated BCDs. The victim became separated from the other two by the current and gave a signal for the others to be collected first. He appeared to look relaxed as he floated comfortably at the surface, but before the tender reached him he was seen to deflate his BCD and submerge. The initial search for him was unsuccessful but he was located later on the sea bed, all equipment in place, with just sufficient remaining air to inflate his BCD to assist raising his body.

Autopsy

The autopsy showed only signs of drowning in a healthy man. There is no evidence that the past injury to his back was implicated in the incident. The cause of death was given as drowning.

Comment

Cause of death was drowning but the reason why this occurred is conjecture. The work of swimming in the strong current plus the choppy surface conditions may have affected his actions. It is possible he pressed the deflate button in error and was too surprised by his submergence to react immediately by dropping his weights, then he inhaled water.

Summary

SCUBA; TRAINED; NO DIVES FOR 4–5 YEARS POST BACK INJURY; APPEARED COMPETENT DIVER; SURFACE SEPARATION BY STRONG CURRENT; BCD INFLATED THEN DEFLATED AND SANK; LOW AIR; FAILED TO DROP WEIGHT BELT; DROWNING.

CASE SC 01/8

An instructor offered to teach five of his friends to scuba dive using hired equipment. He insisted that they first obtain a medical certificate of their fitness to dive. At the time of the first lesson most used the excuse that they could not get an appointment in time, while the 26-year-old victim said he had been examined, which was untrue, but had left the note at home and he would bring it later. After the first lesson, during which he reportedly did not appear to be paying attention, they practised the basic skills in a pool, followed by an open water dive to a maximum depth of 6 msw. The planned duration of the dive was 26 minutes but after 12–13 minutes the victim indicated his need to return to the surface. There he explained he had felt claustrophobic but now felt better and the instructor, who had ascended with him, took him down to rejoin the others and the dive continued uneventfully.

The next day they made their second open water dive at the same location. Water entry was off a sloping rock shelf into shallow water, depth here 9 msw, but they descended initially to about 7 msw. Once again he soon indicated a wish to ascend. At the surface he again reported claustrophobia, but was reassured and he descended with his buddy, the instructor's assistant, to rejoin the others. All proceeded well until he was 'spooked' by a fish and started to make a panic ascent. His buddy grabbed him and tried to calm him but he struggled loose. His buddy just managed to catch one of his fins and, using his own weight, tried to slow the rate of ascent. At the surface, his buddy inflated his BCD for him and responded to his urgent desire to return to land by agreeing to tow him back to the beach. A short time later he "uttered an incredible sound, not a scream", and began making panting or grunting sounds. This was followed by his arms locking, and this muscle spasm lasted till he was only about 10 metres from shore. He was unconscious and not breathing; resuscitation efforts were unavailing.

Examination of the equipment showed damage to both the primary and secondary mouthpieces such that water entry occurred with inhalation. Subsequently, his medical history of bronchitis at age three and seven, prescription of sodium chromoglycate until he was 14, a motorcycle accident in 1998 and a fractured left wrist became available.

Autopsy

A CT scan showed a left-sided tension pneumothorax with a collapsed lung, a smaller pneumothorax on the right side, air in the right ventricle outflow tract but not in the heart, and subcutaneous emphysema. The coronary arteries were healthy. Histology of the lungs showed hypertrophy of the bronchial smooth muscle and basement membrane consistent with a history of asthma.

Comment

When good intentions meet Murphy's Law, the latter wins. Such was the case here. The instructor had not taken all reasonable care when he set aside the non-production of the medical certificates before commencing open water training. He did manage the panic episodes well. Handling any pupil showing panic or claustrophobia requires far more than a black and white decision. Also, the asthma history appears to have been hidden from the instructor. It is not possible to know whether the victim had genuinely forgotten his asthma history or decided to conceal it. The concordance of the latent asthma bronchial sensitivity with the defective mouthpieces producing salt-water aspiration, and his panicked ascent, resulted in him developing pulmonary barotrauma.

Summary

SCUBA; TRAINING CLASS; PANIC EPISODES; ASTHMA HISTORY NOT REVEALED; BORROWED EQUIPMENT; FAULTY MOUTHPIECES ALLOWED INHALATION OF SEA WATER; ADEQUATE AIR; VALIANT RESPONSE BY INSTRUCTOR TO SITUATION; PULMONARY BAROTRAUMA WITH BILATERAL PNEUMOTHORACES.

CASE SC 01/9

This 56-year-old man had an advanced diver qualification and had made a total of 20 dives. He was with his wife, son, and son's girlfriend, and they had signed up for a dive organised by his wife's instructor's dive shop. They were given a pre-dive briefing and had their equipment checked by the instructor before he allowed them to enter the water. His buddy had completed training six weeks previously and was now making his first post-course dive. There was some current and they were advised to snorkel out from the beach to a buoy 15–20 metres offshore before diving, which they did after each added some further weights.

Their contents gauges were reading 100–150 bar when they decided to begin their ascent after the victim had fixed his buddy's loose fin. They were at 10 msw and about two metres apart when they neared a steep rock wall and became separated. The buddy was unable to find him so surfaced in shallow water close to rocks and exited the water. He then heard a shout for him to call an ambulance.

The victim's son was at the surface when he saw his father floating vertically in the rough water close to the rocks, but saw nothing unusual until there was no response to his signal suggesting he join him. When he swam closer he saw his father was now face down and not reacting as the waves washed him over the rocks. He had inflated his BCD although retained his weight belt. His son started to tow him into calmer water and called for help, but nobody heard. The dive class was below them and the son got his father's buddy to descend and get the attention of the instructor. The class was brought up and the instructor helped bring him ashore. Resuscitation efforts were unsuccessful.

Autopsy

A pre-autopsy CT check showed a small amount of air in the right ventricle outflow tract but it was not certain whether this resulted from off-gassing, the vigorous resuscitation efforts, or CAGE. The coronary vessels were healthy. The cause of death was given as drowning.

Comment

The clinical story of the incident suggests CAGE as the most likely cause. It is possible he made a hurried ascent after separating from his buddy, inadequately exhaling as he ascended. The 'advanced open water' certification may have hidden his real inexperience and a lack of confidence. It was noted that he had been under recent work and personal stress.

Summary

SCUBA; 'ADVANCED' DIVER WITH 20 DIVES; BUDDY MAKING FIRST POST-COURSE DIVE; SEPARATION; INFLATED BCD; FAILED TO DROP WEIGHT BELT; PROBABLE CAGE; DROWNING.

CASE SC 01/10

His wife regarded this 65-year-old instructor as healthy but he had confided in his dive buddy that he had recently become short of breath. They had discussed the matter but he did not attend for medical advice. This appeared to be a normal dive until at a depth of about 5 msw he became separated from his buddy. When the buddy surfaced he saw his friend sitting on a rock, gasping for breath. He was brought ashore but collapsed and did not respond to attempts to resuscitate him. He had ditched his weight belt and had plenty of air remaining.

Autopsy

Autopsy revealed severe ischaemic heart disease sufficient to account for death. There was marked coronary atherosclerosis, particularly in the left anterior descending artery, and evidence of a myocardial infarct during the previous six months. There was also some overexpansion of the lungs and it was reported as possible that the decedent had inhaled some water on the return swim.

Comment

This man failed to recognise the significance of his symptoms so did not seek medical advice. He probably left his buddy when first aware of chest or respiratory symptoms and correctly decided to return to the surface.

Summary

SCUBA; EXPERIENCED; INSTRUCTOR; SEPARATION; LEFT WATER BEFORE COLLAPSING; CORONARY ARTERY DISEASE; SUDDEN CARDIAC DEATH.

CASE SC 01/11

This 55-year-old man and his wife had trained 18 months previously and subsequently passed an 'advanced diver' course, but their actual experience is not documented. He was out of character this day, anxious and in an apparent hurry to get started on the dive organised by a friend. Just before they started, a fourth diver joined them and was buddied with his wife, his usual buddy. His wife and her buddy had already descended before he and his friend followed and were unaware of the drama that followed until after they had completed their dive.

Before they descended the buddy made the float with the divers' flag secure, then noticed a fin floating nearby and handed it to the victim, who had apparently not noticed his had come off. While he waited for him to replace his fin the buddy's thoughts were interrupted by a shout warning him someone was in trouble, followed by the command 'inflate your vest'. He then noticed the victim was putting his regulator in and out of his mouth. He quickly inflated both their BCDs and started to tow him to the shore, holding onto his hands. The buddy was badly exhausted when he reached the rocks but helpers there pulled them from the water and started CPR, the witness who had called the warning among them. He happened to be the instructor for the victim's basic training course.

His wife reported that his breathing had been shallower for about nine months, but when he saw his doctor he was told it was due to being overweight.

Autopsy

The autopsy report was that the heart appeared normal and coronary atheroma moderate, maximal narrowing 50% in both right and left main arteries. The left ventricle wall was a normal 15 mm thick. The liver appeared abnormal, with a generalised patchy pallor. The official finding was of cardiac arrest due to coronary artery disease. As there was no formal inquest the police never followed up the widow's request that a report be obtained from the doctor.

Comment

No explanation was known for his behaviour change as he offered none, and no report was obtained from his doctor. His 'automaton' actions with his regulator imply some cerebral dysfunction. The official investigation was terminated as soon as it was found that death was from natural causes. This prevented discussion of the reason for his behaviour, or a request for medical information from his doctor.

Summary

SCUBA; TRAINED; AT SURFACE ABOUT TO DESCEND WHEN BEHAVIOUR CHANGED; BUDDY INFLATED BOTH BCDs AND TOWED HIM TO ROCKS; RECENT BREATHING SYMPTOMS ATTRIBUTED TO OBESITY; CORONARY ARTERY DISEASE; SUDDEN CARDIAC DEATH.

Surface-supply breathing apparatus (SSBA)

CASE H 01/1

This 23-year-old man was employed to dive for crayfish. Because these divers are required to provide their own equipment except for the air compressors and the dories they use, they are regarded as being self-employed and workplace safety regulations are not applied. He held a basic open water scuba certification and had a medical certificate of fitness to dive, so met the requirements for this employment. He was regarded as a careful and experienced diver, and indeed had been given a diver holding both basic and advanced open water certification to train.

The mother boat towed three working dories, the one used by the victim being described as difficult to handle, leaking, and poorly laid out. He was wearing shorts, wetsuit jacket with hood, mask, fins, and a weighted jacket. There was no bail-out bottle. It was the tender's job to follow his bubbles in the dory as he hunted for crayfish. Neither examined the compressor on board properly before diving. After 30 minutes he surfaced 10–15 metres from the dory but descended a short way before the dory could reach him. This ascent and descent was made four times, his gauge later showing he descended 3–6 metres each time, before his last descent. On the last occasion he surfaced he moved his hand over his face making the tender think he was having an equalisation problem like he had the previous day.

This behaviour was strange and outside the tender's experience so it was several minutes before he gave a three-pull recall, using the hose. At this time the compressor stopped but there was adequate air in its reservoir tank so the tender decided to pull the victim up using the hose. When he came into view he was limp, unconscious, and not breathing. There was no response to resuscitation.

Autopsy

The autopsy confirmed that he had been a healthy man. There was no evidence of pulmonary barotrauma, the chest being opened under water to exclude a pneumothorax. There was no air in the heart. Lung histology showed there had been aspiration of food, common in drowning. The carboxyhaemoglobin level was 35%, sufficient to alter his level of consciousness and to lead to confusion and collapse on exertion. The cause of death was given as drowning secondary to acute carbon monoxide (CO) poisoning.

Comment

None of the divers had obtained training as commercial divers because this cost $10,000 and was largely irrelevant to their needs. Open water scuba certification does not include training in maintaining an air compressor or the use of surface-supplied (hookah) breathing apparatus. The equipment had many faults, in particular that the air intake hose kinked easily and had cracks, and the compressor was low in the dory so had reduced air cooling and ran too hot for the lubricating oil used. The jacket weights could not be dropped easily. The absence of a bail-out bottle was not significant in this case as it would have contained CO polluted air. The pressure relief valve was set too low so it was difficult for a diver at depth to obtain enough air.

Summary

HOOKAH; SCUBA TRAINED ONLY; EXPERIENCED; POORLY MAINTAINED HOME-MADE AIR COMPRESSOR; WEIGHT VEST DIFFICULT TO RELEASE; INADEQUATE PRSSURE TO SUPPLY SUFFICIENT AIR AT DEPTH; CARBON MONOXIDE POISONING; DROWNING.

CASES H 01/2 and H 01/3

The air compressor was owned by one of the six friends who took it to a rocky coastal area to hunt for crayfish. Only four planned to dive, and none was either trained or experienced in its use. One was aware of the need to have the inlet upwind but nobody was deputised to supervise its functioning. The first two surfaced after five minutes and complained about the air quality and of headaches. The compressor was moved from the rock hollow onto a pile of stones, so as to be more in the breeze, and two metres of the hose were cut off, stuffed into the intake opening and 'sealed' there with a plastic bag. They then continued their dive till they surfaced again reporting dry mouths. The compressor was now noted to be so hot it had burned the grass near it, so was turned off and the air reservoir vented. There was a single air hose from the compressor and this had a float at the 'Y' junction where the hoses to supply the two divers were attached.

Soon after the next couple of divers entered the water, smoke was seen coming from the air compressor, but this ceased after the air intake was pulled out and the divers continued unaware of this. But 5–10 minutes later the tanks were noticed to be getting hot and someone suggested they pull up the divers. When they came to the surface they were unconscious, and could not be revived.

Autopsy

Pre-autopsy CT examination showed no evidence of arterial gas embolism in either decedent. Both divers had healthy coronary arteries. The carboxyhaemoglobin levels were 28% in H 01/2 and 55% in H 01/3. The cause of death was given as drowning secondary to CO poisoning.

Comment

This double tragedy illustrates the fact that hookah equipment is not a fail-safe alternative to scuba. The two divers would have lost consciousness and drowned without becoming aware of their danger. The level in H 01/3 was approaching the lethal range of CO, while that in H 01/2 was sufficient to cause disorientation or, possibly, loss of consciousness. The compressor unit was home-made and had many potentially lethal factors present, such as loose fittings, and a filter consisting of a nylon bag of cotton wool balls and stocking filled with activated charcoal. Tests of air quality revealed CO and oil levels too high to measure. The divers' toleration of 'dirty air' proved fatal. It is likely that other divers use poorly maintained air compressors and are at similar risk but fail to realise the fact.

Summary

HOOKAH; UNTRAINED; NO EXPERIENCE OF HOOKAH DIVING; HOME-MADE AIR COMPRESSOR WITH MANY MAJOR FAULTS; CARBON MONOXIDE POISONING; DOUBLE FATALITY; DROWNING.

Discussion

BREATH-HOLD DIVERS AND SNORKEL USERS

These fatalities, as usual, fall into two clearly defined groups: those making (or intending to make) breath-hold dives, particularly to spear fish, and those simply swimming while wearing a mask and snorkel. There were six cases in each group. The apparent critical factors clearly differentiated the two groups.

The causes of death in the breath-hold divers were post-hyperventilation blackout drowning in two (BH 01/3, BH 01/12), water power in two (BH 01/5, BH 01/8), trauma (BH 01/6), and acute myocardial infarction (BH 01/10). The danger of hyperventilating to increase underwater duration is well documented but the risk of death from this is sufficiently low to be ignored by those determined to extend their underwater times and disbelieving of the experience of others. Spear fishing is not an altogether safe sport.

The fatal trauma from an outboard motor's propeller resulted from inadequate safety practices on the part of both the victim and the boat driver, and avoidance of such events depends on all parties following recommended safe practices. The acute heart attack was a truly unpredictable event, though had the victim's doctor recognised the true cause of his reported symptoms it is possible he would have been advised not to dive. Water power was the apparent critical factor in two cases, inexperience leading to their inability to correctly manage the conditions. It can be fatal to be in water beyond one's comfort zone.

The other group consisted of swimmers using snorkels, often for the first time, chiefly elderly visitors from out of state. Death among snorkel-swimming visitors to the Barrier Reef is an ongoing problem. While completion of a health questionnaire by boat passengers before permitting them to snorkel is now generally a requirement, it is undoubtedly true that many are unaware of their true medical status. There is also the problem of deciding acceptable levels of risk; to live constantly consulting one's actuarial risk of death may be thought an unhealthy choice. In one case (BH 01/2) the person's obvious anxiety was ignored, and there was failure by those responsible for the safety of the passengers.

The majority of these deaths occurred despite alert safety watchers who may not recognise that a swimmer is in trouble in a crowd when there are no outward signs of a problem, as is often the case. Although one of this group had obtained and worn a buoyancy vest initially, this had been removed before swimming away from the boat. The intended function of these flotation aids is different from that of a life jacket. As these vests tend to float the wearer face down they have a limited safety function in an unconscious wearer, and if they were designed to keep the wearer face up they would not be appreciated by anyone trying to view the marvels of the underwater world! Whether greater stressing of the advice to swim with a buddy would alter behaviour is debatable.

The most common critical factor in this group was cardiac disease with at least three dying from this cause (BH 01/4, 01/7, 01/9) and possibly a fourth (BH 01/1). Simple drowning was the finding in two cases (BH 01/5, 01/11) and here inexperience was a significant factor influencing the course of events.

SCUBA DIVERS

There were 11 fatalities identified in association with scuba diving, of which three (SC 01/1, SC 01/4, SC 01/8), probably a fourth (SC 01/9), and possibly a fifth (SC 01/3) were diagnosable as due to pulmonary barotrauma/air embolism, based on autopsy findings and/or the case history. In two cases (SC 01/1, SC 01/4), the deceased were aware of a looming low-air situation. It is of interest that in case SC 01/4 the victim was accompanied during the ascent and the rate reportedly not excessive. Failure to exhale adequately may occur during an apparently correct ascent. In one case the buddy was unable to fully control the victim's rate of ascent (SC 01/8) and in two cases (SC 01/1, SC 01/9) separation occurred during the ascent. In all of these cases there was a brief delay after surfacing before consciousness was lost. Three of the scuba divers can be considered to have been inexperienced. Claustrophobia has the potential to cause panic and is best not experienced while immersed.

Cardiac factors were thought critical in two cases (SC 01/10, SC 01/11) and in each there had been symptoms mentioned of some occasional breathlessness during the weeks preceding their deaths. It was unfortunate that when SC 01/11 attended his doctor concerning his symptoms his obesity was targeted rather than his heart. Although diver SC 01/3 attended a doctor before her 'refresher' course it is not known whether she revealed either her asthma or history of spontaneous pneumothorax and surgery. Failure by the doctor to keep any notes of the consultation could be regarded as negligent.

The question of the possible significance of a history of childhood asthma in relation to diving safety is raised by cases SC 01/3 and SC 01/8, though in each there were significant additional adverse factors that played a major part in the outcomes.

In the cases where drowning was the given cause of death, the circumstances were unique to each. The dangers of ignoring the 'nanny' advice on safe diving practices is demonstrated, as are the factors of panic and of running low on air. Scuba-related deaths, other than from acute cardiac events, usually show the presence of more than one adverse factor, attention to any one of which would have possibly prevented the fatal outcome. The concordance of several adverse factors supports a contention that the greater the failure to observe the advised safe diving procedures, the less is the margin of safety. This suggests the problem is best tackled through improved training protocols, which should inculcate an intolerance of allowing a low-air situation to develop. Buddy breathing cannot be relied upon as a safe and sure alternative to monitoring the contents gauge, even assuming the gauge is reading accurately. Panic is a killer.

SSBA

Major risk factors are CO in the air through faults in the compressor or positioning of the air-intake hose, and hookah hose disconnection, particularly if no ‘go home’ bottle is worn and no lifeline attached. CO is a silent killer and refusal to accept ‘dirty air’ and imperfectly maintained compressors would be a lifesaver. The lack of enforceable health and safety regulations in the pearling, crayfish, and *beche de mer* industries is a serious concern. Closer controls on the employment of divers appear long overdue. There should be zero tolerance of unsafe working conditions rigorously enforced by all those who oversee workplace safety. It should be mandatory for employed divers to receive formal training in SSBA and diving techniques before using them. It is difficult to see how recreational hookah users can be persuaded of the need for training additional to basic scuba training or the foolishness of tolerating ‘dirty air’.

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Review articles

Decompression sickness in breath-hold diving

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Key words
Breath-hold diving, decompression sickness, review article

Abstract

(Wong RM. Decompression sickness in breath-hold diving. *Diving and Hyperbaric Medicine*. 2006; 36: 139-144.)
Decompression sickness (DCS) in breath-hold (BH) diving has long been disputed as a distinct clinical entity. However, there has been a flurry of case reports describing various symptoms arising in BH divers. Since the 1950s, *Taravana* has been described in French Polynesia, where divers suffered from paralysis, vertigo and nausea. Prior hyperbaric exposure followed by BH dives also led to symptoms and Paulev, a submarine medical officer, described his personal experience of DCS after a series of BH dives. Three similar cases have been described. The Ama divers of Japan and Korea have long been studied, but in the early days no confirmed cases of DCS had been recorded, although Doppler ultrasound had detected circulating bubbles and cerebral injuries have been documented in them. Other BH divers have also been studied, including recreational sports divers and diver fishermen. In all these groups symptoms have been recorded after repetitive deep dives. Theoretical calculations by various authors have concluded that repetitive deep BH dives could give rise to symptoms of DCS. The clinical entity of DCS in BH divers is distinctive in that it tends to affect mainly the brain. Symptoms, even without treatment, tend to subside over time, in many cases without sequelae. Although the aetiological factors causing DCS in BH dives have not been elucidated, there is no doubt that such a clinical entity does exist. Hyperbaric oxygen therapy should be offered to divers with symptoms. Preventive measures are suggested.

Introduction

Breath-hold (BH) diving is common among recreational divers, seafood harvesters, hitherto pearl and sponge divers and, nowadays, competitive freedivers. The risks of breath-hold diving such as barotraumas, salt-water aspiration, near drowning and drowning syndrome, hypothermia, cardiac arrhythmias, marine animal injuries, and ascent hypoxia, 'shallow water blackout', are well known. However, neurological problems caused by decompression sickness (DCS) in BH diving are seldom acknowledged. Does DCS exist as a clinical entity in BH divers?

It has long been held that bubble formation causing DCS from BH diving is most unlikely. Nevertheless, in 1931, Professor Gito Teruoka asserted in his article *Die Ama und ihre Arbeit* that he was aware of the increased nitrogen uptake with increased ambient pressure, but could not find any confirmed cases of DCS among the Ama divers.[1] He indicated then that further research was necessary.

Lanphier believed that bubble formation causing DCS was most unlikely from breath-hold diving, but conceded that this view did not take into account the extremes of depth, frequency and repetition to which breath-hold diving can be carried.[2,3] Using the USN decompression tables, he calculated the probable tissue nitrogen levels for a given series of BH dives and equated them to a specific air-supplied dive for which a decompression schedule or exposure limit could be specified. In his risk assessment, he made the assumptions that the time at depth was spent at maximum depth, ascent and descent were instantaneous, gas uptake and elimination were exponential and the surface/depth (S/D) time ratio was considered.[3] He proposed that no limit of total time needs to be imposed if time at surface is at least equal to time at depth (S/D = 1.0), but that, if the S/D = 0.5, there was a risk of DCS in less than three hours of diving.

Fahlman and Bostrom used a mathematical model to predict nitrogen tensions (PN_2) during hypothetical dives.[4] The model predicted maximum venous PN_2 during a dive to 80 metres of sea water (msw) to be 3 ATA (304 kPa), or 310% higher than the surface equilibrium value (0.74 ATA, 75 kPa). Maximum venous PN_2 during repeated dives to 30 msw were predicted to be 1.44 ATA (145 kPa) and 1.69 ATA (171 kPa) for a surface interval (SI) of 300 and 90 seconds respectively. Predicted venous PN_2 during the first and sixth dive reached 88% and 97% of the maximum estimated venous PN_2 during the entire series respectively. They concluded that the results suggest symptomatic N_2 levels can be reached during BH diving. Furthermore, when diving repeatedly, a SI twice as long as the dive time could help to reduce excessive PN_2 levels.

Nitrogen accumulation measured in brachial venous blood has also been demonstrated in repetitive BH diving in Korean Ama divers, but it was thought that the level of nitrogen accumulation was insufficient to cause DCS.[5] These dives lasted only three hours and to depths of 4 msw.

Evidence of intravascular gas in BH divers

Before the advent of the ultrasound Doppler detector, Schaefer had observed foam in venous and arterial blood drawn immediately after a BH diver surfaced from a single dive to 27 msw lasting one-and-a-half minutes.[6] Subsequent samples drawn ten seconds after surfacing did not show any bubbles, indicating their transient nature. Some ten years later, both Spencer's group and Nashimoto detected venous gas emboli following repeated BH dives in Ama divers.[7,8] Further attempts to identify bubbles in BH divers using continuous Doppler and 2-D echocardiography did not find evidence of circulating air bubbles in BH divers who dived to depths of 24–40 msw over a two- to six-hour period.[9] The tests were performed within an average of 30 minutes of the last dive of the day (range 3–75 minutes). However, bubbles, if present, may have been missed because of their transient nature. Huggins demonstrated Grade I bubbles in a breath-hold diver after a series of repetitive dives in the 46–67 msw range, although in-water oxygen breathing for two periods was given (personal communication, Huggins, 2006). Doppler detection was performed some 18 minutes after surfacing from the last dive.

History

In 1956, Cross alerted the diving community to the existence of *Taravana*, a condition known in Tuamoto Archipelago in French Polynesia where the BH pearl divers suffered symptoms such as vertigo, paralysis, mental anguish and unconsciousness (Table 1).[10] They dived to depths of between 13 and 43 msw with BH times of up to two-and-a-half minutes and surface intervals of between four and ten minutes, usually working a six-hour day. Cross stated that "*Taravana is most frequent toward the end of the day of diving when divers working in a rich lagoon under ideal conditions go crazy with greed and dive until the shell is gone or Taravana strikes*". In Hikueru Lagoon, there were estimated to be 235 divers, who hyperventilated for three to ten minutes, and then descended with the assistance of a lead weight of about 3.5–5.5 kg. At the end of one six-hour day, a total of 47 divers had been affected by various taravana symptoms. However, in nearby Mangareva Lagoon, divers who had 12- to 15-minute surface intervals never suffered from *Taravana*.

In 1957, in Takapoto Lagoon, 43 divers (35 males and 8 females) were observed. There were 13 cases of *Taravana* in a three-week period (one died subsequently) and 12 cases of vertigo and nausea. One was paralysed (paralysis was always accompanied by vertigo, nausea and a general feeling of anguish). Depths of dives were to 15–25 brasses (one brass is approximately one fathom, the length of outstretched arms, a common form of measurement of indigenous divers – about 6 feet or 1.83 metres). In 1958, 34 cases of vertigo, nausea and mental anguish were reported. There were also six cases of partial or complete paralysis, three of temporary unconsciousness with no other symptoms, two were mentally affected and two died. Of the mentally affected, one was unable to recognise his family or home. He was restless, irritable and lacking in understanding of his surroundings. The other was unable to speak coherently even though he seemed normal in other respects.

Symptoms of *Taravana* could last from a few hours to a lifetime. One male Paumotan diver, Tahauri Hutihuti, who was 71 years of age in 1958, claimed he had never had *Taravana*, but he was slow mentally and often missed what he was reaching for. Another diver, Turoa Hutihuti, who was 48 years old in 1958, made frequent dives to 140 fsw for two minutes. He suffered *Taravana* a few times. Once he had paralysis of the right side, and had vertigo and nausea several times; the paralysis lasted three months. He also had a slight visual defect and it was thought this was related to *Taravana*. Paralysis is a common form of *Taravana* and Dr Truc, the French physician, believed more than 95% of it to be temporary, and that the divers would recover completely in a matter of hours or occasionally days.[10]

Cross considered *Taravana* to be a form of DCS, but since no one believed DCS occurred in BH diving, he then considered "anoxia" as a likely explanation. Craig, Lanphier and Rahn had emphasised the danger of hypoxia in BH diving, especially with prior hyperventilation, although they questioned anoxia as an explanation, and suggested that no consequences other than loss of consciousness (LOC) and drowning could occur.[11,12] Craig thought air embolism or DCS could be a likely explanation for *Taravana*.

Reported cases

Paulev experienced symptoms of DCS such as nausea, dizziness and belching, followed by onset of pain in his hip and knee, a weak left arm and tired right arm, as well as paraesthesia and blurring of vision after performing repetitive BH dives to 20 msw for five hours.[13] However, his dives were preceded by a hyperbaric exposure as a chamber attendant for eight minutes at 20 msw. Three similar cases of DCS in BH divers were reported, after they were exposed to pressure in a hyperbaric chamber prior to BH diving.[14]

Bayne and Wurzbacher, as well as Bruch have described cases of pulmonary barotraumas (PBTs) in BH divers who were at depths of 1.8 msw and 4.5 msw respectively.[15,16] Fanton et al reported the case of a spear fisherman who performed 14 repetitive dives per hour for three hours to depths of up to 131 fsw (40 msw).[17] He lost consciousness on surfacing without any evidence of near drowning. Investigation showed abnormal EEG and MRI consistent with focal neurological damage.

Kohshi et al reported multiple cerebral infarctions in two Japanese Ama divers who dived repeatedly between 15 and 25 msw for five hours.[18] Subsequent publications discussed 16 Ama divers, 13 of whom had neurological dysfunction (Table 1).[19,20] In earlier days, Ama divers dived in reasonably shallow waters and also they used to wear cotton suits which precluded them from prolonged stays underwater due to

Table 1
Symptoms experienced amongst three groups of divers (rec. – recreational)

Symptoms	Taravana	Japanese Ama	Spanish rec.
Dizziness/vertigo	34/47	9/16	5/30
Nausea	34/47	6/16	1/30
Motor weakness	6/47	6/16	11/30
Sensory changes		3/16	17/30
Altered consciousness	3/47	1/16	13/30
Headache			13/30
Mental disturbance	36/47	4/16	
Visual disturbance			10/30
Speech disturbance		1/16	7/30
Fatigue			7/30
Motor incoordination			5/30
Memory loss			4/30
Convulsion			1/30
Sphincter relaxation			1/30
Auditory disturbance			1/30
Localised pain			1/30
Cardiorespiratory arrest			1/30
Death	2/47		

hypothermia. These days, however, they wear neoprene suits and make weight-assisted dives, typically with 15 kg weights, and descend to between 15 and 25 msw. Dive times would be between one and one-and-a-half minutes with surface intervals of up to three minutes. They generally work two shifts a day (five-hour working day and five days per week.).

One case described was that of a 33-year-old Ama diver who dived to 22 msw, with a bottom time of one to one-and-a-half minutes with a one-minute surface interval. He commenced diving at 0920 and had 20 minutes for lunch. At 1410 he noted symptoms, which included dizziness and blurred vision in the right visual field. MRI performed four days later showed an infarction in the occipital region. However, his disturbed vision regressed within three weeks. It is interesting to note that in the Ama divers there were no spinal or musculoskeletal symptoms reported. Most symptoms were transient. Some paresis improved within ten minutes. Sensory symptoms, however, took longer to recover, sometimes as long as two to four weeks. Symptoms never appeared on the first day of the diving week, and on the day of injury the Ama diver had dived for at least three-and-a-half to four hours to depths of 20 msw.

Neurological problems have also been reported in competitive sports diving: in multiple constant-weight dives to 25–30 msw; three variable-weight dives to 35–90 msw; and a single no-limits dive to 120 msw.[21 *] These divers suffered from such symptoms as hemiplegia, ataxia, dysarthria, diplopia and colour blindness. The diver who made a single dive to 120 msw had reached that depth several times previously without problems. On this occasion, however, he used a new assisted-ascent technique (at 4 $msw.sec^{-1}$). Shortly after surfacing, he experienced paraesthesia in the right leg, followed within minutes by a right-sided hemiplegia that responded to recompression treatment within 30 minutes using a US Navy (USN) Treatment Table.[22]

A large number of Spanish spear fishermen using submarine scooters for BH diving have suffered neurological symptoms. They have managed to achieve depths of 25 to 46 msw, with BH times of up to four minutes (90 to 240 sec, mean 133 sec; Table 1).[23] A depth of 63 msw has also been reported, the number of dives varied between 15 and 20 dives per hour over a period of three to eight hours. The surface interval was usually two minutes or less. Symptoms were immediate on surfacing and were all neurological (Table 1).[24]

In Australia, spear fishing is popular among BH divers. They usually perform multi-day diving spending 5–6 hours in the water each day. Depths of dives range from 13–20 msw to 27–30 msw. Dive times are usually in the vicinity of two to three minutes with a one-minute surface interval; sometimes with deeper dives, a two-minute SI would be common. A preliminary unpublished survey by the author indicated that very few of those who suffer symptoms consult their medical practitioners. This is due mainly to the lack of appreciation that DCS could occur in BH diving and also the fact that such symptoms are usually attributed by the divers to other causes such as viral illness. Furthermore, symptoms are normally of short duration, and mostly no sequelae are experienced.

Symptoms arising from breath-hold diving

The symptoms encountered among different groups of BH divers are diverse and vary in frequency (Table 1). The most common include dizziness, ataxia, nausea, hemiparesis, paraesthesia, visual and speech disturbances and altered states of consciousness. Symptoms common in scuba divers such as musculoskeletal pains and spinal cord involvement are uncommon in BH divers unless there has been previous hyperbaric exposure prior to BH diving. Musculoskeletal pain has been reported in only two cases: one in an Australian BH diver (two days after the event) and the other in a Spanish recreational diver.[13] In eight Australian spear fishermen, a similar range of symptoms to those listed in Table 1 were noted, headache, nausea and dizziness being the most common.

Lesions, particularly in the Japanese Ama divers, tend to be centrally located with sparing of the spinal cord or musculoskeletal systems. Some of the symptoms described by BH divers could have been due to nitrogen narcosis (such as euphoria), hypoxia (altered level of consciousness,

* Footnote: For definitions see Mckie N. Freediving in cyberspace. *SPUMS J*. 2004; 34: 101-3.

muscular weakness and incoordination, loss of motor control, visual disturbance), carbon dioxide retention (headache, dizziness, confusion and amnesia) or even middle/inner ear barotraumas or alternobaric vertigo (vertigo, nausea, disorientation, visual disturbance) rather than to DCS.

As already stated, even without treatment most of the symptoms in BH divers subside spontaneously. The Japanese Ama divers recorded 10 minutes to four weeks, the Spanish divers took 30 minutes to 72 hours and the Australian spear fishermen took 12 to 36 hours. In the *Taravana* cases, in whom paralysis was the most common symptom, more than 95% completely recovered in hours, the rest in days.

Common factors causing symptoms

Factors causing neurological symptoms include:

- dives in excess of 20 msw
- repetitive dives of three hours or more
- a rapid rate of ascent by necessity
- surface intervals of short duration, shorter than the depth time.

However, it has been reported that a BH diver who performed repetitive dives for three-and-a-half hours to depths of only 8 msw developed severe headache, dizziness, blurred vision, vertigo, numbness and weakness of all four limbs on surfacing and had to be rescued.[25] No clinical evidence of pulmonary barotrauma was detected. Although the presumptive diagnosis was CAGE, it could also have been hypoxia leading to loss of motor control (LMC).

Lindholm and Lundgren have shown that 11% of competitive BH divers performing static apnoea had symptoms of hypoxia such as LOC or LMC.[26] Two subjects who had LMC had P_AO_2 of 19.6 and 21 mmHg (2.6 and 2.8 kPa) respectively.

Mechanism

The aetiological factors of DCS in BH diving have not been elucidated. As noted previously, the Ama divers of Mishimi Island of Japan never complain of symptoms on the first day of the diving week. Their symptoms when manifested appear only after at least three-and-a-half to four hours of repetitive diving to depths of 20 msw and when the surface interval is less than the depth time, suggesting nitrogen accumulation could be a contributing factor. We do not know how long it takes for the body to totally eliminate excess nitrogen from repetitive diving. In compressed-air diving, the USN decompression tables assume a 12-hour surface interval to be clear of residual nitrogen time, whereas the DCIEM tables assume an interval of 18 hours. It is feasible that it takes much longer than 18 hours. After repetitive dives to 3 ATA, sufficient nitrogen is absorbed to cause supersaturation. Fahlman and Bostrom have concluded that symptomatic N_2 levels could be reached during repetitive BH dives using their mathematical model to calculate hypothetical dives.[4] Based on computer modelling, Olszowka and Rahn calculated that N_2 accumulation occurs in fat tissue increases throughout repetitive BH dives, but brain PN_2 does not increase with repetitive BH dives, preventing clinically significant autochthonous bubble formation in the brain.[27]

It is assumed that bubble formation occurs in the venous circulation based on Doppler evidence.[7,8] The lungs have such an efficient filter that the bulk of the bubbles are prevented from reaching the left side of the circulation. However, on repetitive dives, some of the bubbles could reasonably bypass the lung filters and reach the arterial circulation.[28,29] Buoyancy then assists the bubbles to reach the cerebral circulation.

Various other mechanisms of DCS have been postulated, such as:

- Cardiac shunting via an atrial septal defect (ASD) or patent foramen ovale (PFO). However, evidence from the Ama divers surveyed does not lend support to PFO or ASD as a contributing factor.[18]
- Pulmonary barotraumas. These lead to CAGE and are not common in BH diving.[14,15]
- Bubble formation in the arterial circulation. Although arterial bubbles have been seen in decompressed animals, bubbles are unlikely to form *de novo* in large arteries. Inert gas supersaturation sufficient to provoke bubble formation is improbable in arterial blood since the healthy lung essentially equilibrates alveolar and arterial gas tensions in a single pass. Arterial supersaturation may occur in a very rapid ascent of 6 $msw.sec^{-1}$, but arterial bubbles have proven difficult to demonstrate even under these conditions.[30]
- Microbubbles.[31,32] Bubbles smaller than 21 μm can pass through capillaries in the brain and do not usually cause any lesions.[33] However, Hills and James have shown experimentally that microbubbles can impair the blood-brain barrier transiently.[34] It is possible that with repetitive diving, microbubbles can and do impair the cerebral circulation, as has been seen in the Ama divers who show multiple cerebral infarctions in the terminal or border zone of cerebral arteries visible on MRI.[17,19] Such lesions could be the consequence of microbubbles which are too small to be detected by conventional Doppler technique.

Treatment

Most BH divers do not appreciate that BH diving could cause DCS and consequently when they have symptoms do not seek medical advice. Nonetheless, even without treatment, symptoms tend to subside and generally have no sequelae. No standard protocol of treatment has been agreed upon. BH divers have received surface oxygen and various hyperbaric treatment tables have been employed, which include USN 6, USN 5, Comex 12, Comex 18, HBO 14.[21,23] Adjuvant therapy such as nifedipine, non-steroidal anti-inflammatory drugs, heparin, steroids and diazepam have all been tried.

Divers who present with symptoms after BH diving should be offered hyperbaric oxygen therapy. A delayed presentation is not a contra-indication to treatment. A BH diver presenting four days after his dives with symptoms of tiredness and cognitive dysfunction, and cerebellar signs and failed sharpened Romberg test, as well as poor short-term memory, responded to a USN 6.[35]

Prevention

It would appear that to avoid DCS from BH diving, the following empirical strategy could be adopted:

- dive no deeper than 20 msw (although this might not be practicable)
- limit the number of dives per day or dive less than three hours continuously
- ensure the SI is at least twice as long as the dive time
- if feasible, breathe surface oxygen, at least during the lunch break and at the end of the diving day
- the Ama divers could conceivably breathe oxygen to decompress for 5 to 10 minutes at 6 msw at the end of the diving day.

It has been claimed that oxygen decreases decompression time by 30% to 50% depending on the depths of the dives. Imbert and Bontoux, using the French air decompression tables with in-water oxygen decompression, indicated that oxygen decompression not only saves decompression time but also has the effect of decreasing the incidence of DCS to two to three times lower than air decompression for dives of the same depths and bottom times.[36]

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This article is based on a presentation by Dr Wong at the Symposium on Breath-hold Diving, Orlando, Florida, hosted by the Undersea Hyperbaric Medicine Society and Divers Alert Network, 2006.

SPUMS Annual Scientific Meeting 2007

Dates: April 15 - 20
Venue: Oceans Resort, Tutukaka, Northland, New Zealand

Guest Speaker
Neal Pollock, PhD
Themes
From mountain high to ocean deep – the physiological challenges of extreme environments
Medical aspects of technical diving

Neal Pollock is a research physiologist at the Center for Hyperbaric Medicine and Environmental Physiology, Duke University Medical Center, Durham, NC. He is also heavily involved in DAN International and was one of the editors of the recent guidelines for scuba diving and diabetes. He works regularly in Antarctica and has also been involved in high-altitude physiology studies. He thus brings a wealth of expertise to our meeting and is an excellent speaker.

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Obesity and diving

Gisele ML Mouret

Key words
Obesity, decompression sickness, medical conditions and problems, health status, review article

Abstract

(Mouret GML. Obesity and diving. *Diving and Hyperbaric Medicine*. 2006; 36: 145-7.)
Obesity has long been accepted as one of the many risk factors for developing decompression sickness (DCS) when diving. Whilst body fat is a great heat insulator, there are a number of disadvantages to an obese person diving. These disadvantages range from the physical restriction of wetsuits, to concurrent medical pathologies and the higher demands on the cardiovascular and pulmonary systems whilst swimming. The high solubility of nitrogen in lipids and fats results in 5.4 times as much storage of nitrogen in fat as muscle tissue. This increases supersaturation of fat with nitrogen and significantly increases the risk of developing DCS. Whereas, historically, dive medicals precluded obese people from diving, a review of the literature would suggest that a sensible approach to diving and the medical examination can minimise risks of DCS to the obese patient.

Introduction

Decompression sickness (DCS) is a disease that arises as a consequence of bubbles forming within tissues.[1,2] Inert gases, such as nitrogen and helium, enter the body through the lungs during inspiration. At depth, and therefore under pressure, gas dissolves in the blood and is carried to the tissues into which it diffuses.[3] The process is repeated in reverse as gas is transported back to the lungs for exhalation.[4] It is widely held that when a state of supersaturation occurs during decompression, the tension of dissolved gas exceeds ambient pressure, and bubbles form.[1,2] In 1880 Paul Bert found that the DCS bubbles are composed almost completely of nitrogen and a small amount of water vapour.[5] The amount of nitrogen it takes to saturate a diver will depend on the diving depth and individual characteristics of size and body composition.[2] Many factors, including obesity, may be associated with the development of DCS.

Nitrogen bubbles form in watery tissues (blood, muscle) and fatty areas (subcutaneous fat, peri-articular and para-spinal fat).[5–7] Nitrogen is 5.3 times more soluble in fats/lipids than in water.[5,8] Blood supply to adipose tissue, however, is poor, and the release of nitrogen from adipose tissues is slow.[6,9]

The main advantage of obesity in divers is the tendency for adipose tissue to be a good energy source and an excellent insulator.[8,10] In comparison with the overwhelming risks to obese divers, however, this advantage fades into insignificance.

Obesity

Whilst body mass index (BMI) is regarded as one of the best predictors of adiposity, individual physique must be taken into consideration and, on occasion, skin fold measurements taken to indicate percentage body fat.[6,10–12] The BMI is calculated by dividing the individual's weight (kg) by the square of the individual's height (m) .[5,10] Obesity is generally accepted as a BMI greater than 30 kg/m^2.[5,11]

Obesity and DCS

Increased BMI implies an increased fat content of the body. This increased body fat leads to increased nitrogen storage and hence possible excessive nitrogen bubble formation and thus increased risk of development of DCS.[2,5,7,8,12,13]

As early as the mid-nineteenth century, it was observed that "corpulent" caisson workers were more likely to suffer from DCS than others, whilst on the basis of animal research in the early 1900s, it was recommended that plump men be excluded from high-pressure caissons. In a group of 932 caisson workers, the odds ratio for DCS in obese versus non-obese men was 2.2 (CI 1.3–3.9, $P < 0.01$).[14]

US Navy divers with the highest quartile skin fold thickness (20% overweight) have been reported to have an increased risk of developing DCS.[6,12] Those 25% or more overweight have been estimated to have a tenfold increased risk of developing DCS.[7,15] On average, DCS in USN divers was experienced by overweight compared with lean individuals.[7] Other USN studies have been inconclusive, possibly because few obese divers were included in the study groups. It has been suggested that increased age leads to increased risk of DCS, but that with increased age there is a tendency towards increased adiposity and decreased fitness.[1,13,14] Altitude studies have also suggested that increased body weight was associated with an increased risk of DCS in aviators.[16] Overall, it is difficult to establish clearly the influence of obesity on the incidence of DCS, since DCS is an uncommon event with multiple variables and most studies were poorly designed and ingrained with diving dogma.

Dive computers are increasingly relied upon by recreational divers. These dive computers are programmed with dive tables that are calculated for the average-sized individual and do not take into consideration obese divers.[2,9,13]

Understandably, therefore, the use of dive tables by obese divers is also increasing their risk of developing DCS.

Obesity and fitness

Obesity often implies a decrease in exercise tolerance and poor physical fitness.[1,2,6] The increased cross-sectional area of the obese diver has been interpreted to mean a larger workload due to additional 'drag' through the water.[8] Whilst poor exercise tolerance implies impaired ability to self-rescue, there are also implications as to the obese person's ability to perform 'buddy' duties.[6] It has been postulated that poor aerobic fitness is a possible cause of increased individual susceptibility to DCS.[13] Additionally, aerobically trained individuals have a lower risk of developing DCS because they have a lower risk of developing venous bubbles. Broome et al (1995) found that aerobically trained pigs are less likely to develop DCS than a control sample of untrained pigs.[2]

Obesity and co-morbidities

Obesity is often found to co-exist with other medical problems. In the diving environment, these co-morbidities can be exacerbated, or lead to the development of new problems. At the extreme, co-morbidities can result in an increased chance of sudden death. Obesity itself can cause the individual to have developed ventricular enlargement/hypertrophy.[17] Likewise, the increased size of the individual increases the circulating blood volume and thus the cardiac output will have increased. Carbon dioxide (CO_2) retention, left ventricular dysfunction and hypoxaemia can all lead to increased pulmonary artery pressures.[17] Additionally, cardiac arrhythmias may exist and worsen when the individual dives.

Obesity increases the incidence of ischaemic heart disease and therefore increases the risk of a cardiac event.[8,11] Hypertension is common in the obese and there is a greater mortality rate in the hypertensive obese than in lean counterparts.[11,17] Hyperlipidaemia is also associated with increased body fat.[7,11] Hypertension and hyperlipidaemia are known risk factors for cardiac events.[7]

Pulmonary disease is found in about a quarter of the obese.[17] In addition to this, the physical changes associated with excess weight restrict movement and increase cavity pressures. Excess adipose tissue surrounding the chest wall reduces compliance and forced vital capacity. Increased abdominal pressure reduces functional residual capacity (FRC) and increases dead space, thus leading to increased pulmonary shunt. This results in increased CO_2 retention and hypoxia.[11,17] An adjunct to this is an increase in workload which itself increases oxygen consumption and CO_2 production.

Diabetes mellitus is a known co-morbidity of obesity. Swings in glucose levels during the stress and exertion of diving may increase the risk of drowning.[11] There are also increased rates of gastro-oesophageal reflux disease, which in turn offers its own risks to diving.

Obesity and medications

Many appetite suppressants have psychotropic effects and their CNS interactions with nitrogen under pressure are unknown. They may also elevate blood pressure thus potentially increasing cardiovascular stress.

Obesity and dysbaric osteonecrosis (DON)

The possibility that obesity poses an increased risk of developing DON has been raised.[10,18] The incidence may vary according to the conditions of the hyperbaric exposure. Risk factors include the degree of obesity, number of dives, depth, decompression profile, and rate of compression. In the 1970s the rate of DON in Japanese fishermen reached 50–60% and was attributed to adiposity.[18] Research has found a significant incidence of DON in obese mice compared with thin mice.[19]

Obesity and immersion

Immersion offers its own effects on the human body. The water pressure alone moves approximately 700 ml of blood from the periphery into the thorax. This vascular congestion reduces elastic recoil of the lungs and reduces FRC.[17] The blood pressure and cardiac output will increase causing a diuresis and thus a decrease in blood volume.[17] Surrounding water pressure also exerts a force against the rib cage, reducing chest wall compliance, and abdomen, causing elevation of the diaphragm and thus further reducing FRC.[17] Upon descent, gas density increases. This, combined with the reduced chest-wall compliance, may lead to the obese diver experiencing a large increase in the work of breathing.

The diving wetsuit will impose further restrictions on the obese diver. As the wetsuit compresses the skin, cutaneous blood is shunted centrally exacerbating the already congested lungs. Additionally, the wetsuit will restrict chest wall movement, further decreasing compliance and increasing the work of breathing.[17]

The obese person needs a larger wetsuit increasing buoyancy, which means that the obese person needs to wear a considerably heavier weight belt.[8] Due to the lower level of fitness, the obese person uses more oxygen when diving, therefore they either have shorter dive durations or need to use larger tanks. The combined effect is that the obese diver may have limited ability to rescue themselves or their buddy and difficulties accessing the dive launch from the water.[8]

Current protocols

Some authors have called for obesity to be a contra-indication to diving (> 20% excess weight).[6,12] There are, however, some advantages to the obese being able to exercise in a weightless environment.[8] A less harsh view, with the aim of minimising risks on an individual basis, may be a better way to proceed.[6] A thorough medical examination is advocated, including fasting blood tests and exercise tolerance testing. A sensible approach to diving is also recommended. This includes an attempt to improve cardiovascular fitness, and introduce slower ascent rates, fewer dives in 24 hours and a reduction of bottom time by 25–50%.[1–3,8,9,13,15] Reducing cardiovascular load by avoiding diving in strong currents and tides is also suggested.[7] With these guides implemented and a good general understanding of the risks involved, the obese diver can minimise poor outcomes.[3,6]

It has been suggested that new, safer tables be developed incorporating current knowledge of physiology. The challenge has been set to improve models "to cover more of the people more of the time".[3]

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This article is based on a presentation by Dr Mouret at the SPUMS Annual Scientific Meeting in Fiji, 2006.

Explosion in Peruvian chamber

A 65-year-old patient of the Hyperbaric Clinic in San Borja, Peru, was incinerated recently after a powerful explosion in a monoplace oxygen chamber. An unauthorised camera in the chamber was thought to be the cause, but the Clinic would not comment. The explosion, which blew in the door and windows of the surrounding room, occurred minutes after the patient entered the chamber. According to criminology experts, death occurred in a matter of seconds. Alfonso Gonzales, a representative of the Peruvian Hyperbaric Medicine Society, said that this centre did not have a registered doctor or technicians. "We had already warned the Ministry of Health and other institutions of this fact", said Dr Gonzales. Hyperbaric medicine has been practised in Peru for ten years.

Editor's comment: This case reaffirms yet again the need for meticulous management and the highest safety standards, combined with properly trained staff, in hyperbaric facilities.

The diving doctor's diary

Case report. Flying after diving

Carl Edmonds

Key words
Case reports, decompression sickness, barotrauma, flying (and diving)

Prologue

I have previously used SPUMS case reports as a forum for illustrating new or poorly recognised clinical features of diving disorders. I have taken the liberty of using this report as a teaching aid/discussion case. Some details have been deliberately altered to avoid embarrassment to certain colleagues. My dictatorial attitudes have been incorporated in italics.

SB was an enthusiastic and experienced diver who had rapidly progressed through the certification system and was well qualified to undertake deep or prolonged salvage diving, using dive computers. Nevertheless, he tried to stay just within no-decompression limits, because of apprehension regarding decompression sickness (DCS), which he had experienced in the past. Unfortunately in this instance he did require decompression from an energetic multi-level salvage dive. The maximum depth was 40 metres' sea water (msw) and it was a repetitive dive.

During the 5 msw stop he was feeling a bit tired and was developing a deep central headache. He had noticed this on a previous dive, but it had cleared up without incident. This time it progressed during the final ascent and was severe after reaching the surface.

As he was diving on a Pacific Island about three hours' flight time from Australia, he contacted an Australian diving emergency system and explained the dive parameters and his symptoms – headache, tiredness and a "fuzzy feeling" in his head.

Transport was immediately available, so after an hour or so on oxygen (O_2), which seemed to impart some relief, he was flown to Australia on a commercial flight. During the flight, on which he was not allowed oxygen, his headache became more severe.

Surface oxygen: *After a decade or two's delay, the USA and UK experts have now indisputably validated the 1970s Australian/French advocacy of this regime. However, prudence is still required. A previous diver medevac from the Cook Islands to Tahiti, resulting in the explosion of the plane with the death of all on board, was possibly attributable to a higher than normal cabin oxygen percentage.*

On landing, he was taken to a recompression chamber (RCC) and given an extended US Navy Treatment Table 6 (USN 6), with good results. It was extended when the symptoms seemed to recur during the final ascent from 9 msw to the surface.

As he had no residual clinical features, he was permitted to return to New Caledonia one week later.

Diagnosis: acute decompression illness

Terminology: *I think they meant DCS. I have some difficulty with the various inclusions and exclusions of the "decompression illness" appellation. I do not think they were implying cerebral arterial gas embolism, but who is to know when terminology is so ill defined.*

All went well until he was being flown back, when the headache recurred in the aircraft. He arrived in Noumea with symptoms almost identical to those he had started with (perhaps a little less severe). The whole procedure was then repeated with another medevac and USN 6 recompression treatment in Australia.

The prognostic advice was then complicated by the presumed diagnoses. The clinical features were interpreted by some as indicating a serious and acute (neurological) DCS. As such, the recommended delay before further aviation exposure was up to two months, with a permanent exclusion from diving. Most of his advisers, however, suggested a couple of weeks before flying (especially considering his aviation-induced recurrence), and a month or two before diving.

Flying after DCS treatment: *If RCC treatment with 100% O_2 has been adequate, no further bubbles will remain, and the tissues will be effectively de-nitrogenated, so it is difficult to understand how altitude exposure can aggravate the basic pathology of DCS. This is especially so if the treatment has been instituted without air breaks (this adds more nitrogen to bubbles and tissues) and if the asymptomatic diver has breathed 100% O_2 intermittently on the surface post-treatment for a number of hours.*

To be ultra-conservative, and to avoid the possible aggravating factors of mild hypoxia, alkalosis and hypocapnoea associated with commercial aviation, an oxygen mask could be used during the flight, with allowance made for adequate ventilation.

If, however, the RCC treatment has been inadequate to completely remove the gas phase from the tissues, then the duration before safe flight will be proportional to the incompetence of the treatment. Some recompression facilities advise weeks' or months' delay before aviation exposure.

He consulted me because of the conflicting advice regarding an appropriate time delay before resumption of flying and diving; a reasonable concern considering his repeated experience and the varying advice. As I considered his oxygen recompression therapy initially to have been more than adequate, and as it was given by competent therapists (i.e., with a well-fitting oxygen mask and for a considerable duration), I decided to consider other possibilities.

A more detailed history revealed previous headaches associated with diving, some episodes of mild sinus barotrauma of descent, and the use of 'negative pressure' middle ear equalisation techniques. The ENT system seemed problematic, but there was no obvious non-diving ENT pathology.

After performing sinus CT scans, I advised him to return to Noumea by boat, and to change his middle ear equalisation techniques from the Toynbee/swallowing types to the conventional Valsalva, commence equalisation on the surface (pre-descent), and perform it more often (every metre or so of descent). The CT scan, performed within three days of the 'recurrence' and subsequent RCC 'treatment' revealed moderate mucosal thickening especially affecting the sphenoidal sinus.

Radiology and scanning: *This CT scan ideally could be replaced by MRI. CT and MRI have made sinus X-rays obsolete because of the explicit pathology that can now be demonstrated. Significant mucosal swelling is easily demonstrated with either scanning technique, but even with X-rays the pathology was well demonstrated in the past – if the positioning was accurate – and showed the frequency of the sphenoid pathology with diving, either alone or with other para-nasal sinus involvement. Always think 'sphenoid or ethmoid' when investigating diving-induced headaches.*

Scans often return to normal a week or two after sinus barotrauma. That is why the consultant otologists, who see patients 'cold', due to delayed referral and investigation, are at a disadvantage and may offer inappropriate reassurance. Had I been more courageous, I might have sent him home by air with advice to repeatedly perform Valsavas every minute during ascent and descent, but there was no way of assuring that the sphenoidal sinus was adequately patent, or that the advice would have been conscientiously followed.

Para-nasal air spaces: *Some techniques of middle ear equalising involve negative nasopharyngeal pressures – tending to induce swelling of the mucosa and narrowing of the Eustachian tube and sinus ostia. Other factors being equal, positive pressure techniques, such as the Valsalva, are less likely to be associated with barotraumas of descent, and the subsequent ascent complications. Physicians have often been deceived into assuming that middle ear equalisation affects only the middle ears. It affects the whole naso-pharynx and may be very relevant to sinus equalisation when the ostia are only marginally patent.*

Follow-up correspondence revealed an uneventful trip home and a successful return to diving, now employing appropriate equalising techniques.

Had the sphenoidal barotraumas not been prevented by this simple alteration in middle ear auto-inflation technique, some may have advised surgical intervention. I would not have recommended sphenoidal ostial endoscopy as a treatment (because of its potential complication rate), although I would have done so had the maxillary sinus been the one involved. This reflects a somewhat conservative attitude, as I am less concerned with operative complications in the maxillary than in the other sinuses.

Medical literature: *The literature on sinus barotrauma in diving is less than comprehensive, but experience at a diving medical clinic at the Great Barrier Reef suggests that it is a relatively common complaint. Aviators have described some reasonable clinical series of sinus barotrauma ("aerosinusitis") but the only two series on divers that were more than individual case reports were published in this journal. One involved 50 'hot' cases as they presented post-dive at the RAN School of Underwater Medicine and the other had 50 'cold' cases, a more clinically severe group, referred for treatment to the Diving Medical Centre. All these, and other relevant literature references, are to be found in the chapter on sinus barotrauma in* Diving and subaquatic medicine.[1]

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Articles reprinted from other sources

Breathing performance of ‘Octopus’ demand diving regulator systems [Abstract and executive summary]

Anthony TG, Fisher AS, Gould RJ

Abstract

Self Contained Underwater Breathing Apparatus (SCUBA) often use an ‘Octopus’ system as an alternative air supply. QinetiQ at Alverstoke were contracted by the Health and Safety Executive (HSE) (Contract D5008) to conduct a review and breathing performance test of ‘Octopus’ systems. It was shown that SCUBA single demand valve systems capable of meeting the breathing performance requirements of BS EN 250, cannot be relied upon to meet the same requirements when used as part of an ‘Octopus’ system. Reduced breathing performance of ‘Octopus’ systems (when compared to single valve systems) was found to be a result of the use of low performance first stage regulators, second stage demand valves of different and poor performance and breathing in phase as opposed to out of phase. Recommendations on the use of ‘Octopus’ systems are presented. Appropriate test procedures and acceptance criteria should be identified for ‘Octopus’ systems, and proposed for the next revision of BS EN 250.

Executive summary

When using Self Contained Underwater Breathing Apparatus (SCUBA) it is recommended to use an appropriate alternative breathing gas source/secondary life support system. ‘Octopus’ systems are often used to fulfil or support this requirement.

BS EN 250:2000 specifies the performance requirement of a single demand valve, first stage regulator combination. This, however, gives no indication as to how an ‘Octopus’ two demand valve, first stage regulator combination might perform.

The Centre for Human Sciences at QinetiQ Alverstoke was contracted by the Health and Safety Executive (HSE), Contract D5008, to conduct a review and breathing performance test of ‘Octopus’ systems.

A literature review was conducted. Based on data available from the review and in consultation with the HSE, six configurations of ‘Octopus’ systems were selected and purchased anonymously for test. The selections sought to emulate purchases likely to be made by UK divers.

The systems were evaluated for compliance with elements of BS EN 250 and the Norwegian Petroleum Directorate/UK Department of Energy guidelines for breathing apparatus, when used both as single demand valves and in tandem as ‘Octopus’ systems. The pass/fail criteria adopted encompassed both BS EN 250 and the NPD/DEn guidelines.

Test data obtained showed that SCUBA single demand valve systems capable of meeting the breathing performance requirements of BS EN 250 cannot be relied upon to meet the same requirements when used as part of an ‘Octopus’ system.

Reduced breathing performance of ‘Octopus’ systems (when compared to single valve systems) was found to be a result of the use of low performance first stage regulators, second stage demand valves of different and poor performance and breathing in phase as opposed to out of phase.

The observed breathing performance of ‘Octopus’ systems may go some way to explaining the number of divers who inexplicably break contact with their buddies during alternative air supply (AAS) ascents using SCUBA ‘Octopus’ systems.

The results support the view that the preferred system for an alternative air supply is a completely independent gas supply and demand regulator.

If ‘Octopus’ systems are to be used it is recommended that:
- Divers are made aware that although CE marked valves to BS EN 250 may be considered as ‘fit for purpose’ when used alone, their performance cannot be assured when configured as part of an ‘Octopus’ system.
- Octopus systems should be based on a high performance first stage regulator.

- Octopus systems should be configured with demand valves of similar performance.
- Older valves, or valves whose performance may have degraded should not be used.
- The diving community should be made aware of the effects of breathing in and out of phase.

Appropriate test procedures and acceptance criteria should be identified for 'Octopus' systems and proposed for inclusion in future diving apparatus standards, including the next revision of BS EN 250.

QINETIQ Ltd, Alverstoke, Building 4, Room 1, Fort Road, Gosport, UK

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Key words
Reprinted from, scuba, equipment, performance, emergency ascent, diving safety memos

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The evidence-basis of diving and hyperbaric medicine. A synthesis of the high-level clinical evidence with meta-analysis [Abstract]

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Abstract of the thesis submitted for the degree of Doctor of Medicine of the University of New South Wales
Satisfied requirements on 23 May 2006

Abstract

Introduction: Hyperbaric oxygen therapy (HBOT) is the administration of 100% oxygen at pressures greater than 1 atmosphere. One recurrent criticism that has been made of this field is that treatment is based on little or no good clinical evidence.
Aims: The primary objective of this thesis is to make a useful response to that criticism. I planned to collate all the available randomised evidence in the fields of diving and hyperbaric medicine, supply a critical appraisal of each paper, and synthesise that evidence in a series of systematic reviews with meta-analysis. I also intended to use a cost analysis of hyperbaric practice in our own facility to inform formal cost-effectiveness analysis using the estimates of effect generated by the individual meta-analyses.
Methods: A comprehensive search strategy was used to identify all clinical RCTs involving the administration of hyperbaric breathing mixtures. Each trial was appraised using the software developed by the Oxford Centre for Evidence Based Medicine. Each critical appraisal was loaded onto a searchable website at <www.hboevidence.com>. Each diagnostic category identified was considered for inclusion in a Cochrane systematic review and meta-analysis.
Results: The database includes 130 critical appraisals covering 173 separate reports. The site has received more than 17,000 hits. There are 12 formal meta-analytical reviews and all have been accepted for publication in the Cochrane Database of Systematic Reviews at the time of writing. These form the basis of this thesis and include late radiation tissue injury, chronic wounds, acute hearing loss and tinnitus, multiple sclerosis and decompression illness. The meta-analyses in this thesis suggest there are several areas where HBOT is associated with improved clinical outcomes and that routine use is probably justified in some areas (e.g., radiation proctitis healing with HBOT: NNT 3, 95% CI 2 to 11). On the other hand, these analyses suggest there is most unlikely to be significant clinical benefit from the application of HBOT to patients currently referred for HBOT (e.g., multiple sclerosis).
Conclusions: The randomised evidence for the use of HBOT is now significantly easier to access. Recommendations for therapy and future research directions can be made on the basis of these analyses.

Key words
Reprinted from, underwater medicine, hyperbaric oxygen therapy, medical conditions and problems, research, evidence, Cochrane library

The database of randomised controlled trials in hyperbaric medicine maintained by Dr Michael Bennett and colleagues at the Prince of Wales Diving and Hyperbaric Medicine Unit is at:

<www.hboevidence.com>

Management of mild or marginal decompression illness in remote locations workshop proceedings

[Final consensus statements, editorial notes and executive summary]

Simon J Mitchell, David J Doolette, Christopher J Wacholz and Richard D Vann (editors)

Final consensus statements

Consensus statement 1

With respect to decompression illness (DCI), the workshop defines "mild" symptoms and signs as follows:

- limb pain[1,2]
- constitutional symptoms
- some cutaneous sensory changes[3]
- rash

where these manifestations are static or remitting[4,5] and associated objective neurological dysfunction has been excluded by medical examination.

Footnotes

1 The workshop agrees that severity of pain has little prognostic significance, but acknowledges that severity of pain may influence management decisions independent of the classification of pain as a "mild" symptom.

2 Classical girdle pain syndromes are suggestive of spinal involvement and do not fall under the classification of "limb pain."

3 The intent of "some cutaneous sensory changes" is to embrace subjective cutaneous sensory phenomena such as paraesthesiae that are present in patchy or non-dermatomal distributions suggestive of non-spinal, non-specific, and benign processes. Subjective sensory changes in clear dermatomal distributions or in certain characteristic patterns such as in both feet, may predict evolution of spinal symptoms and should not be considered "mild."

4 The proclamation of "mild" cannot be made where symptoms are progressive. If the presentation initially qualifies as mild and then begins to progress, it is no longer classified as "mild" (see also Footnote 5).

5 The possibility of delayed progression is recognised, such that the "mild" designation must be repeatedly reviewed over at least the first 24 hours following diving or the most recent decompression, the latter applying if there has been an ascent to altitude. Management plans should include provisions for such progression.

Consensus statement 2

The workshop accepts that untreated mild symptoms and signs[1] due to DCI are unlikely to progress after 24 hours from the end of diving.[2]

Footnotes

1 Mild symptoms and signs are strictly limited to those defined in Statement 1 and its footnotes.

2 This statement does not hold where there is a further decompression, such as further diving or ascent to altitude, in the presence of mild symptoms.

Consensus statement 3

Level B epidemiological[1] evidence indicates that a delay prior to recompression for a patient with mild DCI[2] is unlikely to be associated with any worsening of long-term outcome.

Footnotes

1 Levels of evidence in American Family Physician [Internet]. [Leawood(KS)]: American Academy of Family Physicians; c2004 [Cited 2004 Dec 6]. Available at: <http://www.aafp.org/x17444.xml>

2 "Mild DCI" is limited to those presentations exhibiting only "mild symptoms and signs" strictly as defined in Statement 1 and footnotes.

Consensus statement 4

The workshop acknowledges that some patients with mild symptoms and signs after diving[1] can be treated adequately without recompression. For those with DCI, recovery may be slower in the absence of recompression.

Footnote

1 The non-specific reference to "mild symptoms and signs after diving" is intentional. It reflects the fact that the manifestations may or may not be the consequence of DCI. The statement suggests that even if they are the result of DCI, full recovery is anticipated irrespective of the use of recompression although resolution may take longer. Importantly, "mild symptoms and signs" are strictly limited to those defined in Statement 1 and footnotes. Where symptoms and signs fall outside the spectrum of manifestations herein defined as "mild," standard management and therapy is indicated.

Consensus statement 5

The workshop acknowledges that some divers with mild symptoms or signs[1] after diving may be evacuated by commercial airliner to obtain treatment after a surface interval of at least 24 hours, and this is unlikely to be associated with worsening of outcome.[2,3,4]

Footnotes

1 "Mild symptoms and signs" are strictly as defined in Statement 1 and footnotes.

2 It should be noted that most favourable experience with commercial airliner evacuations comes from short-haul flights of between 1 and 2 hours' duration. There is much less experience with longer flights.

3 It was agreed that provision of oxygen in as high an inspired fraction as possible is optimal practice for such evacuations. In addition, the risk of such evacuation will be reduced by pre-flight oxygen breathing.

4 It was emphasised that contact must be established with a receiving unit at the commercial flight destination before the evacuation is initiated.

Editorial notes

Given the title of the workshop and proceedings, the reader who peruses these statements without a full appreciation of the discussion that led to their final wording may be confused by the absence of specific reference to remote locations. During the consensus discussion it became clear that ethical and legal concerns could be minimised if guidelines for important management decisions were applicable irrespective of the patient's location. Care was taken to make this so, and the consensus statements therefore do not specifically refer to DCI in remote locations. It is acknowledged, however, that the environmental and logistic characteristics of a remote location (such as weather, aircraft availability or material condition) may need to be considered in management decisions in the interests of patient safety, irrespective of the guidelines promulgated here.

The statements are self-explanatory, but the reader should note that some of them are heavily qualified with footnotes. These qualifications are non-negotiable components of the meaning of each statement, and the statements should not be quoted without reference to these footnotes. Of particular importance is the strict definition of mild symptoms and signs in Statement 1 and footnotes. All references to "mild" in the subsequent statements are linked back to this definition. It follows that statements 2–5 should not be quoted without reference to Statement 1.

Statement 4 is perhaps the pivotal outcome of the workshop. Its intent requires contextual explanation so that the concerns and commentary of the workshop participants are accurately reflected. The statement supports a decision not to recompress for mild symptoms and signs (as defined) after diving where, for example, there is suspicion the symptoms may not be caused by DCI, or where there are logistic or safety reasons to avoid evacuation, such as might exist in a remote location. Statement 4 also reflects the workshop consensus that if the symptoms are due to DCI but they fit the "mild" criteria, then medium to long-term disadvantage to the patient is very unlikely if they are not recompressed. This is clearly quite different from a directive that "henceforth, all cases of mild DCI do not require recompression." Statement 4 should not be interpreted in this way. Statement 4 merely notes that some patients are unlikely to be disadvantaged by not being recompressed and provides the treating clinician with options for sensible decision making according to the prevailing circumstances. The word "some" is used intentionally to indicate that it is the clinician's final decision whom to recompress or not. The statement cannot be generalised to allow treatment funding providers to make funding policy decisions about recompression for all mild DCI.

A statement acknowledging the practice of in-water recompression was discussed but not included in the proceedings. The rationale for this deletion was the primary workshop focus on mild DCI. In view of the earlier determinations, especially Statement 4, in-water recompression was not an option likely to be pursued for patients whose presentation met the criteria for "mild" DCI signs and symptoms. In-water recompression was endorsed as an option for severe remote DCI management during the evolving clinical problem evolution (see hypothetic problem discussion), but no policy statements were generated. Its deletion from the consensus statements should not be interpreted as rejection of its utility. The reader is referred to the proceedings of the UHMS in-water recompression workshop for more information.[1]

Similarly, an attempt to provide a consensus statement describing an appropriate time interval between recompression for DCI and flying, usually for the purposes of returning home, was rejected due to insufficient data. There was general agreement that more work is needed in this area.

Reference

1 Kay E, Spencer MP, editors. In-water recompression. *Proceedings of the 48th Workshop of the Undersea and Hyperbaric Medical Society*; 1998 May 24; Seattle. Kensington (MD): Undersea and Hyperbaric Medicine Society; 1999. 108pp.

Executive summary

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Decompression illness (DCI) results from the formation of bubbles in body tissues during reduction in ambient pressure. Bubbles can affect any organ system, and DCI may be diagnosed following the onset of one or more characteristic manifestations following a compressed gas dive. Severe manifestations of DCI typically develop rapidly following diving and include central neurological symptoms and signs and, more rarely, cardiopulmonary collapse. The nature and latency of mild symptoms of DCI are more variable. Typical symptoms are limb pain, constitutional symptoms, rash and sensory changes without central neurological manifestations. Since bubbles can be detected in the blood following most dives, and since divers frequently experience vague symptoms following diving, the boundary between “mild DCI” and “no disease” is indistinct.

First-aid treatment for DCI centres on oxygen breathing to accelerate the washout of other gases from bubbles and tissues. Definitive treatment of DCI is recompression to reduce the size of bubbles, with hyperbaric oxygen breathing to accelerate the washout of other gases. Hyperbaric oxygen also has therapeutic actions independent of bubble resolution. Recompression therapy is particularly efficient when administered within minutes following diving; presumably early bubble dissolution limits pathophysiology. Even if recompression is not immediately available, it is self-evident that delay should be minimised for central nervous system DCI and additional decompression (such as by unpressurised air flight) should be avoided during transport to recompression facilities. This viewpoint has guided the management of DCI of all severities for nearly a century.

The purpose of this workshop was to evaluate the precept of urgent, pressurised evacuation for recompression in the context of mild symptoms of DCI and recreational divers amongst whom delay to recompression is typically greater than 20 hours. This was motivated by the increasing popularity of recreational diving in remote locations where even emergency air evacuation to recompression facilities will take many hours. The only present source of data is from retrospective analysis of databases containing cases of mild DCI where treatment has been delayed. Analysis of such Level B1 evidence[1] (epidemiological data not derived from high-quality randomised controlled trials or systematic reviews) risks biased estimates of prognosis or effect of interventions. Specific difficulties with these databases are that they are likely contaminated with non-cases owing to the diagnostic ambiguity for mild DCI and the only outcome measure is the presence or absence of residual symptoms following treatment.

Mild symptoms of DCI that are static or remitting at 24 hours after diving are unlikely to progress to serious symptoms. No incidents of such deterioration were found in databases from several large treatment centres and several recreational and naval databases. Also, divers often do not seek treatment for mild symptoms, and there is no evidence of consequent long-term health problems in the recreational diving population.

The existing literature is divided on whether delay to recompression for DCI influences the treatment outcome, and some of this ambiguity is due to an interaction of disease severity and urgency. However, for mild symptoms of DCI, delay does not appear to influence long-term outcome. Careful filtering to remove doubtful diagnosis from DAN data (1987–97) revealed that delays longer than 12 hours resulted in 5.9 per cent incidence of residual symptoms at the end of all recompressions compared to 3.9 per cent for shorter delays. Delay was a less potent predictor of outcome than other factors (e.g., age), and there was no difference in percentage of divers with complete relief at three, six, and nine months.

Aeromedical evacuation of a diver is costly, can be logistically difficult, may not result in a clinically relevant reduction in delay to recompression, and is not without risk. These might seem to outweigh any potential benefit in mild DCI. Such conventional cost-benefit analysis is dependent on the integrity of any diagnosis of mild DCI and is clouded due to cultural and social expectation of standard of medical care, and because the cost is usually borne by a third party. Informed risk assessment by the diver in a remote location with mild DCI requires a strong doctor–patient relationship that is unlikely to exist, leaving the doctor to make this decision.

Private ground transportation, usually without supplemental oxygen or intravenous fluids and a typical delay to treatment of 42 hours, is the most common form of retrieval to the recompression facilities in Townsville that service the majority of the Great Barrier Reef. There was no difference in the incidence of residual symptoms at the end of all recompressions between 80 divers with mild symptoms of DCI retrieved by ground transport and 22 divers retrieved by air (typical delay 24 hours).

Where logistics determine the necessity, divers with mild symptoms of DCI can make short-haul flights aboard commercial airliners without any apparent influence on subsequent recompression treatment outcome. DAN data (1998–2002) contained 1,108 divers with pain or mild neurological symptoms of whom 95 flew with symptoms before recompression. There was no significant difference in the incidence of residual symptoms at the end of all recompressions between divers with mild neurological symptoms who did not fly and those who flew more than 24 hours after diving; however, there was a significantly higher incidence of residual symptoms in divers who flew sooner than 24 hours. There were no such differences for divers with pain as their only symptom.

Treatment options in remote locations include non-recompression therapies that should be employed as first aid during any delay to recompression and may be sufficient treatment alone for mild symptoms. Standard non-recompression therapies are based on known pathophysiology and include 100 per cent oxygen breathing (see above), fluid replacement to reduce haemoconcentration, and drugs to reduce platelet activation. Inflammation is probably an important aetiology of mild symptoms, and more trials are needed of non-steroidal anti-inflammatory drugs, antihistamines, and possibly emerging anti-inflammatory therapies. Options for hyperbaric oxygen treatment that may exist in remote locations include recompression chambers not staffed by experienced diving medical officers or in-water recompression. Choice of these options would depend on evaluation of the patient, facilities and staff.

There is a perceived risk of relapse from flying soon after treatment. Current recommendations for delay before flying following treatment are not evidence based and range from three days to six weeks. Amongst 95 divers surveyed by DAN, there was no difference in the rate of relapse (9 per cent) between divers who flew earlier or later than three days following treatment.

As a prelude to producing a consensus statement, two hypothetical cases, one of serious DCI and one of mild DCI, were used to stimulate discussion of workshop issues amongst a panel of experts. These discussions are supplemented by one subjective account of lessons learnt during 25 years of treating DCI in remote locations. The discussions illustrated that no standardised management algorithm could replace clinical judgement. However, there was unanimous support for hypothetical case management that did not require aeromedical evacuation or recompression therapy for a patient with mild symptoms after diving in whom the diagnosis of DCI was equivocal.

The workshop consensus statements generated in regard to these issues are presented at the beginning of the workshop proceedings.

References

1 Levels of evidence in American Family Physician [Internet]. [Leawood(KS)]: American Academy of Family Physicians; c2004 [Cited 2004 Dec 6]. Available at: <http:/ /www.aafp.org/x17444.xml>

Key words
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An analysis of marine animal injuries presenting to emergency departments in Victoria, Australia [Abstract]

Taylor D McD, Ashby K, Winkel KD

Abstract

Objective: To describe the epidemiology of marine animal injury in Victoria, Australia, in order to identify risk factors and recommend prevention strategies.
Methods: Retrospective, descriptive study of patients with marine animal injuries who presented to Victorian emergency departments between October 1995 and June 2000. Data were obtained from the Victorian Emergency Minimum Dataset. The main outcome measures were the marine animal involved; the nature, time, and place of injury; and subject demographics and activity.
Results: Two hundred five injuries were identified, and males predominated (71.7%, $P < 0.01$). Injuries were most frequent during summer and when jellyfish were most prevalent. Various fish species, stingrays, jellyfish, and sharks were incriminated in 83 (40.5%), 46 (22.4%), 42 (20.5%), and 5 (2.4%) injuries, respectively. Most (65.9%) injuries occurred during leisure or sport, and 72 (35.1%) occurred in a place of recreation. Spikes, spines, and barbs caused 82 (40.0%) injuries, and stings caused 54 (26.3%) injuries. Bites were uncommon. Most injuries were to the limbs, with the hands or feet injured in 127 (62.0%) patients. Forty (19.5%) injuries were associated with a retained foreign body. Only 17 (8.3%) patients required admission to the hospital.
Conclusions: Marine animal injury is seasonal but rarely serious. Vigilance is required when handling fish, and protective gloves, footwear, and clothing are recommended where appropriate. Clinicians should consider retained foreign bodies in penetrating injuries. Warnings are recommended when jellyfish are most prevalent.

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Reprinted with kind permission from Taylor D McD, Ashby K, Winkel KD. An analysis of marine animal injuries presenting to emergency departments in Victoria, Australia. *Wilderness Environ Med*. 2002; 13: 106-12.

Key words
Reprinted from, marine animals, injuries, envenomation, toxins, epidemiology

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Features of glossopharyngeal breathing in breath-hold divers [Abstract]

Seccombe LM, Rogers PG, Mai N, Wong CK, Kritharides L, Jenkins OR

One technique employed by competitive breath-hold divers to increase diving depth is to hyperinflate the lungs with glossopharyngeal breathing (GPB). Our aim was to assess the relationship between measured volume and pressure changes due to GPB. Seven healthy male breath-hold divers, age 33 (8) [mean (SD)] years were recruited. Subjects performed baseline body plethysmography (TLC_{PRE}). Plethysmography and mouth relaxation pressure were recorded immediately following a maximal GPB manoeuver at total lung capacity (TLC) (TLC_{GPB}) and within 5 min after the final GPB manoeuver (TLC_{POST}). Mean TLC increased from TLC_{PRE} to TLC_{GPB} by 1.95 (0.66) litres and vital capacity (VC) by 1.92 (0.56) litres ($P < 0.0001$), with no change in residual volume. There was an increase in TLC_{POST} compared with TLC_{PRE} of 0.16 (0.14) litres ($P < 0.02$). Mean mouth relaxation pressure at TLC_{GPB} was 65 (19) cmH_2O and was highly correlated with the per cent increase in TLC ($R = 0.96$). Breath-hold divers achieve substantial increases in measured lung volumes using GPB primarily from increasing VC. Approximately one third of the additional air was accommodated by air compression.

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Reprinted with kind permission from Seccombe LM, Rogers PG, Mai N, Wong CK, Kritharides L, Jenkins OR. Features of glossopharyngeal breathing in breath-hold divers. *J Appl Physiol.* 2006; 101: 000-000. First published May 11, 2006; doi:10.1152/japplphysiol.00075.2006.

Key words
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ANZCA Annual Scientific Meeting 2006

Abstracts from the Diving and Hyperbaric Medicine Special Interest Group session

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Hyperbaric oxygen for osteoradionecrosis prophylaxis – treat at 203 or 243 kPa?

Michael Heytman and David Wilkinson

Purpose of study: Hyperbaric oxygen (HBO) is currently used in the treatment of a variety of radiation tissue injuries. HBO is also used to prevent the onset of mandibular osteoradionecrosis (ORN) when planned surgery is undertaken on the previously irradiated mandible; this is termed ORN prophylaxis. A tested protocol involves 20 HBO sessions pre-operatively and 10 sessions post-operatively. While this number of exposures is widely used, HBO has been applied at a range of treatment pressures – typically 243 kPa (2.4 ATA) or 203 kPa (2.0 ATA). Arguments exist for each; however, there is no clinical evidence favouring one over the other. This institution has, over a period of time, treated ORN prophylaxis at both of these pressures. Is there any difference in outcome between these two treatment pressures for our facility?
Methods: With institutional ethics committee approval, an audit was undertaken for patients treated with HBO for ORN prophylaxis between 1992 and 2004. Retrospective case note review documented treatment pressure and whether healing was achieved following surgery.
Results: 38 cases providing the required information were found. For 10 cases treated at 243 kPa there was one case of new ORN (failed prophylaxis). The range of HBO was 12–33 treatments with the case of ORN receiving an atypical course of 12 HBO treatments. For the 28 cases treated at 203 kPa there were two cases of ORN with a range of 29–32 HBO treatments. There was no difference in risk of ORN for these two groups (two-tailed Fisher Exact test, $P = 1.0$).
Conclusions: For the small sample analysed, no difference in outcome could be found for mandibular ORN prophylaxis treated with HBO at either 243 kPa or 203 kPa. Our experience does suggest there is a risk for failed prophylaxis if the published protocol of 20 HBO pre-surgery and 10 HBO post-surgery sessions is not followed.

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Key words
Meetings, osteoradionecrosis, hyperbaric oxygen therapy

Bleomycin and hyperbaric oxygen therapy – what is a rational approach based on the current evidence?

Ian Dey

Bleomycin is a chemotherapeutic agent used for the treatment of squamous cell, testicular and lymphomatous cancers. Interstitial pneumonia and chronic pulmonary fibrosis are therapy-limiting adverse effects of bleomycin. Case reports exist of patients previously treated with bleomycin being exposed to hyperoxia during general anaesthesia subsequently developing fatal respiratory distress syndrome. Largely as a result of these case reports previous bleomycin exposure is considered by many as an absolute contra-indication to hyperbaric oxygen therapy. It is this author's belief that bleomycin should be considered a relative and not an absolute contra-indication to hyperbaric oxygen therapy. Small numbers of patients previously treated with bleomycin have been uneventfully treated with hyperbaric oxygen and many have successfully undergone general anaesthesia. To make a rational risk-versus-benefit decision we must consider in detail what the current evidence suggests in regards to the general risks of bleomycin and oxygen therapy and whether it is possible to risk stratify patients previously treated with bleomycin, identifying those at high risk of an adverse outcome. Factors such as time lapsed since bleomycin treatment, the total dose, evidence of pulmonary complications and the patient's creatinine clearance all need considering. We must also of course consider how strong the indication for hyperbaric oxygen therapy is. This review attempts to make sense of evidence available in an effort to provide clinicians with a rational approach should a patient previously treated with bleomycin be referred to Hyperbaric Medicine.

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Key words
Meetings, hyperbaric oxygen, toxicity, medications

Iatrogenic arterial gas embolism in Australia – a demographic perspective

Margaret B Walker

Arterial gas embolism (AGE) is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation. Air may enter the arteries directly (arterial air embolism (AAE)), or may enter via the venous system (venous air embolism (VAE)), passing into the arterial circulation through right-to-left shunts, or by overwhelming the filtering capacity of the pulmonary circulation. Bubbles passing through the cerebral circulation cause endothelial damage, with resulting cerebral oedema due to capillary leak, and multiple areas of local ischaemia due to arterial occlusion.[1] There have recently been a number of articles on the topic of gas embolism in the medical literature and to those of us working in the field of Hyperbaric Medicine, it is surprising that there is still little understanding of the condition in the general medical community, in particular the role of hyperbaric oxygen therapy (HBOT).[2,3] This presentation will concentrate on AGE resulting from iatrogenic causes, and attempt to define the demography of iatrogenic AGE in Australia, based on data collected at the major hyperbaric medicine units over the past 10 years.

A total of 39 cases of iatrogenic air embolism were seen and treated in Australian hyperbaric medicine units in the last 10 years. In approximately half of these the arterial gas embolism occurred during cardiac surgery. Most were diagnosed post-operatively after a relatively uneventful bypass procedure, when the patients manifested neurological signs, most commonly hemiparesis or focal seizure activity on awakening. Some were treated on the basis of a witnessed event in which a large amount of air was seen to pass into the circulation during the operation. The remaining cases had a varied aetiology, most commonly radiological procedures (lung biopsy, coronary or cerebral angiography), laparoscopic surgery, hysteroscopy, and air entrainment into venous access lines. There was one case of massive helium embolism following direct inhalation of helium from a gas cylinder at a party, and one blast injury.

The most common treatment regime for AGE in Australia is the Royal Navy Table 62, with extensions as required, followed by daily oxygen treatments at 2.8 or 2.4 ATA as needed. Lignocaine was used as an adjunct to HBOT in 66% of cases. The time delay between injury and treatment with hyperbaric oxygen ranged from one hour to several days. Sixty-two per cent were treated within six hours, with two-thirds of these patients gaining a complete recovery. Overall, 60% of patients had a complete recovery to normal neurological function, and a further 15% were significantly improved, regardless of delay to treatment.

Benefits of HBOT for treatment of gas embolism include elimination of gas by establishment of a diffusion gradient, reduction in bubble size as a direct result of increased ambient pressure, prevention and treatment of cerebral oedema, and reduced leukocyte adherence to damaged endothelium.[4] Immediate recompression produces the best response, but there is growing evidence that delayed HBOT is still beneficial, especially in AAE.[5]

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Key words
Meetings, cerebral arterial gas embolism (CAGE), hyperbaric oxygen therapy

Post cardiac-surgical strokes – a role for HBO?

Alastair J Gibson

Introduction: Post-operative strokes occur in a small percentage of adult cardiac surgical patients and have devastating consequences for these patients. There is evidence to suggest that cerebral arterial gas embolism (CAGE) is an important aetiological factor in most of these cases. Hyperbaric oxygen therapy (HBOT) is the administration of 100% oxygen at greater than atmospheric pressure. It is accepted as the definitive treatment for CAGE related to scuba diving accidents. The similarities between this and the pathophysiology of post cardiac-surgical strokes due to iatrogenic CAGE suggest that potential benefits from HBOT may accrue to these patients.
Purpose: The purpose of this study is to review the experience of treating post cardiac-surgical stroke patients in our local hyperbaric facility, including their presentation, delay before treatment and outcomes. The current evidence base for treating such patients is reviewed.
Methods: A retrospective case series analysis was conducted.
Results: Over a 10-year period, patients with post cardiac-surgical strokes and who presented within the first 48 hours were referred for HBOT, of whom 12 were treated. The neurological outcomes were excellent in all but one case who died. A review of the literature provides a rational basis for the potential benefits of HBOT in this scenario but at present there are only limited clinical data to support its use.
Conclusions: The postulated mechanisms for the development of post cardiac-surgical strokes provides a sound theoretical basis for the suggestion that the use of HBOT is associated with improved outcomes. However there are no prospective randomised data to support such a claim. Such a trial would be problematic and, until more evidence is available, HBOT should be considered on a case-by-case basis.

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Key words
Meetings, cerebral arterial gas embolism (CAGE), hyperbaric oxygen therapy

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BSAC approves 12-year-old diving members

The minimum diving age for branch members of the British Sub-Aqua Club has been lowered from 14 to 12, bringing the rules into line with those of its commercial arm, BSAC Schools International. Of club members who responded to a postal vote, 945 voted for the reduction, 504 against.

BSAC's National Diving Committee is preparing training guidelines for 12- to 14-year-olds. Under the BSAC Schools system, youngsters below 14 can train only to BSAC Ocean Diver, a starter level with a maximum diving depth limit of 20 m.

Regarding children's danger-awareness and dive-management abilities, [Clare] Peddie's [BSAC National Diving Officer] view was that they should understand issues in broad terms, while being assured of safety under water by diving under the close supervision of suitably qualified adult guardians. She did not think the physiological effects of diving on still-developing teenage bodies presented a dilemma. "*Quite a lot of research on children in diving has been done by CMAS and PADI, and there's no evidence to suggest that the young are any more susceptible to decompression illness than adults*," she said.

Extract taken from *Diver magazine*, July 2005

Obituaries

Victor Brand, MC

GP, Anaesthetist
Born: 16 July 1914
Died: 9 June 2006

Victor Brand, who was awarded the Military Cross for his bravery and devotion to treating injured soldiers in the open during a ferocious battle against the Japanese in Malaya in January 1942, has died of pneumonia at Bethlehem hospice in Caulfield. He was 91.

Victor was the second of five children of Isaac and Toba, who migrated from Poland to Melbourne via Palestine in 1913. He was educated at Caulfield Grammar, and graduated in medicine from Melbourne University in 1937. Victor became a resident at The Alfred Hospital and then worked as a locum around Victoria and the Riverina. In his first locum stint, he had to perform a tonsillectomy on the patient's kitchen table.

Victor joined the army in 1940 and, as Captain Brand, he became the Regimental Medical Officer of the 2/29th Infantry Battalion. Before sailing for Singapore, he married Emilie "Fifi" Fry in June 1941. She had arrived from Marseilles, France, in the early 1930s. From January 1942, Fifi had no definite news of her husband, who was listed as missing in mid-1942, until she was advised in January 1943 that he was a POW in a Malayan camp. He was a prisoner of the Japanese for 3.5 years and continued to administer treatment to other POWs, both at Changi and on the Thai side of the infamous Burma-Thailand railway.

In September 1943 it was confirmed that Victor had been awarded an MC for his heroic deeds during the Battle of Muar 10 months earlier. Under continuous bombardment and disregarding his own safety, he remained in the open, tending wounded Australian and Indian troops. When the battalion withdrew he stayed with the wounded and loaded them into trucks, which were subsequently stopped at a roadblock. He was then forced to leave behind those who could not walk, escorting the walking wounded through jungle to the battalion. The 2/29th and the 2/19th battalions had held up the advancing Japanese for six days to enable the main British force to pull back to avoid being trapped. Nearly 60 per cent of the 2/29th were killed and the wounded men left in the trucks were later massacred by the Japanese at Parit Sulong. Victor never got over the slaughter of these men.

Later, on the Thai-Burma railway, Victor spent nearly a year operating a primitive hospital for up to 200 men in a rainforest clearing 240 kilometres up the line from Bampong in Thailand. (Edward "Weary" Dunlop was providing similar selfless service on the Burma side of the railway.) Victor's only supplies at first were a wok, machete, bamboo and quinine tablets. Most of the men suffered from malaria, dysentery and cholera. "*I cut down on a vein in the ankle and inserted a length of bamboo – with half the bore of a drinking straw – attached to lengths of stethoscope tubing fixed to the bottom of a pint-sized container*," Victor wrote of his treatment of cholera victims. "*The idea was to pour about six pints of boiled and strained river water with added rock salts into the patient's bloodstream in 24 hours.*"

With the liberation of Singapore in August 1945, he went down to the docks every day. After a meal on a British battleship, he got back to Changi "*waving a slice of white bread, the first seen for over three years,*" he wrote. He also wrote of a hospital ship moored at the wharf. "*Outside in the dusk was a long line of emaciated men dressed in a few rags. I could see them enter into the light and be divested of their rags and mess tins and taken away to be washed. They returned dressed in pyjamas and were put to bed between the white sheets, so tenderly.*"

His time in Changi and in Thailand, he was always at pains to point out, was nothing compared with the suffering endured by the men building the railway – in fact Changi was relatively comfortable, except for the shortage of food. Years later, he visited the memorial at Changi and, on seeing hundreds of white crosses, wept uncontrollably.

Back in Melbourne, Victor initially practised as a GP and later as an anaesthetist. He served as an honorary at the Alfred from 1951 to 1971. Although opposed to Australia's involvement in Vietnam, in 1966 he joined a voluntary group of medical specialists and nurses, sponsored by the Alfred, to serve in a civilian hospital near Saigon. Victor was always a passionate supporter of Israel and, following the Six-Day War in 1967, he volunteered to work in a military hospital.

He loved scuba diving with an underwater camera or spear fishing around Aireys Inlet. As a founding member of SPUMS, he dived in many exotic locations, including Fiji, Vanuatu, Tahiti and the Solomon Islands. He had a wonderful memory, a powerful intellect and a sharp sense of humour. He devoured books of all kinds and in his last year re-read classics by Anatole France and the complete volumes of Voltaire in French. On Anzac Day this year he proudly led the 8th Division AIF again as one of the last surviving decorated officers. He is survived by his children, Melanie and Andrew, and three grandchildren.

Andrew Brand, Andrew Kennon and Melanie Brand

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Key words
Obituary, personality, reprinted from

Victor Brand, friend and colleague

Victor Brand and I joined the Grey Street Anaesthetic Group at about the same time in 1965. I had come to Melbourne from the country, while Victor had been in solo practice in Melbourne for many years. He was a wonderful colleague and friend. It was only after SPUMS was founded and an advertisement appeared in the *Medical Journal of Australia* that he mentioned that he had been a diver for many years and would like to join SPUMS. At that time I was the Medical Officer for the Royal Australian Naval Reserve diving team in Melbourne.

He had some interesting stories of dramatic events. One was while diving in the Red Sea with friends on board a yacht. This was in the days before contents gauges had become common and long before we met. Victor found that nothing came from the regulator when he tried to breathe in and started for the surface. Later he could not remember what had happened, but was told that he arrived at the surface and appeared to be unconscious, so was hauled onto the boat where he woke up to see worried faces all around. By the time he had finished lunch he no longer felt tired so he, like someone thrown from a horse, decided to show that he was able to dive again. This time the dive went swimmingly. He and I never worked out whether he had had an air embolism or went unconscious from dilution hypoxia. Whatever the diagnosis, the result was a very much on-the-ball Victor. His first pair of flippers, which died at a SPUMS ASM in Truk, were from France. They were slip-on black rubber sandshoes with a narrow pocket on each side. The rods that held the paddle part of the fin fitted into these pockets. During a dive one pocket tore and the paddle part swung free. Victor was a bit disappointed as the fins were getting close to voting age, in those days 21 years old.

I remember the time when Victor told me that he would stop diving in Port Phillip because after an October dive he had been shivering and blue with cold when he climbed out of the water. When I said “You’ll have to get a thicker wetsuit,” his reply was something like “I’ve always only worn bathers when diving”. However, he did get a wetsuit for diving with SPUMS.

Victor and Fifi were regulars at SPUMS ASMs and were friends with everyone. He took an active part in SPUMS for many years, asking intelligent questions during scientific sessions and offering good advice around the bar.

John Knight

Key words
Obituary, personality

Gareth Long

Gareth was born on 11 March 1967 in a British Military Hospital at Terendak near Malacca, Malaysia, where his father was the Regimental Medical Officer to an infantry battalion. He died on 20 August 2006 in a tractor accident at his property in Linkwood. He was found by a neighbour after the overturned tractor was observed.

Gareth eventually arrived in Australia in August 1971, via Germany, with his parents, Geoff and Ann, and two sisters, Xanthe (who also died tragically, aged 16 years) and Kerri. After being educated locally Gareth spent two years at Knox Grammar School in Sydney where he learnt the meaning of life! Then it was a year’s working holiday around Australia in his trusted VW beetle – the forerunner to the Porsche in which all dashing young registrars should carouse.

Then it was off to medical school and the Prince of Wales Hospital, Sydney. It was during these years that many friendships were cemented and much beer consumed. Gareth graduated as an orthopaedic surgeon in 2001 and worked in many rural locations around NSW, including Bega. Here, with his infectious charm and enthusiasm, he led the campaign for the building of a new hospital. Gareth’s skills and ability were better suited to Canberra; however, his roots remained in Bega where he had purchased Thornhill, a small grazing property. On this land he built a magnificent home where he hoped to one day settle and raise a family; an ideal base from which to continue his skiing and scuba pursuits. This latter interest brought him into contact with Society members, who will miss his talents, charm and wit. Those of us who attended the annual meetings with both Gareth and his father will never forget the bond that they shared, father and son – the best of mates. Many of us have had the privilege of receiving examples of his skilled underwater photography, wonderful keepsakes of time spent together.

Generous to a fault, Gareth supported the Bega Rural Fire Brigade, the local football club and travelled widely donating his time and expertise to those less fortunate. These hearts-and-minds expeditions most recently took him to Bali where he both taught the local surgeons and operated himself. At the time of his death, Gareth, through World Vision, was supporting four children. Perhaps it was these children that he was thinking of, or his own children in the future, whilst he attacked the black wattles to improve the pastures at Thornhill on the tractor that senselessly took him from us.

Gareth is survived by his parents, Geoff and Ann, his sister and brother-in-law, Kerri and Anthony Dowd, nephews Cody, Sebastian and Oliver and his very special friend, Jaana Giannos of Canberra. The family has suggested, if Society members wish to remember Gareth, that donations in his name to World Vision would be appreciated.

Hamish Turnbull

Key words
Obituary, personality

SPUMS notices and news

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 medically qualified, and be a financial member of the Society of at least two years' standing.
2 The candidate must supply evidence of satisfactory completion of an examined two-week full-time course in Diving and Hyperbaric Medicine at an approved Hyperbaric Medicine Unit.
3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4 The candidate must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, and in a standard format, for approval by the Academic Board before commencing their research project.
5 The candidate must produce, to the satisfaction of the Academic Board, 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, and obtain instructions before submitting any written material or commencing a research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. 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 NHMRC/AVCC statement andguidelines on research practice (available at http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm) or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documented evidence of approval by an appropriate research ethics committee. It is expected that the research project and the written report will be primarily the work of the candidate.

The Academic Board reserves the right to modify any of these requirements from time to time. The Academic Board consists of:
Dr Fiona Sharp, Education Officer, Professor Des Gorman and Associate Professor Mike Davis.

All enquiries should be addressed to the Education Officer:
Dr Fiona Sharp,
249c Nicholson Road
Shenton Park, WA 6008
Australia
E-mail: *<sharpief@doctors.org.uk>*

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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Extract of ratified minutes of SPUMS Executive Committee Telephone Conference Meeting held on 12 March 2006

Opened: 0908 hr

Present: Drs C Acott (President), R Walker (Immediate Past-President), S Sharkey (Secretary), D Smart (ANZHMG Representative), D Vote (Committee Member), C Lee (Committee Member) (Dr Vote excused at 0936)

Apologies: Drs M Davis (Editor), G Williams (Treasurer/Public Officer)

1 Minutes of the previous meeting
Ratified.

2 Matters arising from the previous minutes
2.1 Contracting of the SPUMS Administrator is being progressed.
2.2 SPUMS Committee overseas representatives have been sent an e-mail to confirm their continuing availability. Action ongoing.
2.3 In response to their request the ANZCA SIG has been offered a position on the SPUMS education board for authorisation of the SPUMS Diploma award to ANZCA certificate candidates. A response remains outstanding.
2.4 The following motions will be put forward at the 2006 AGM:
2.4.1 Constitutional amendments enforced by Consumer Affairs.

2.4.2 Motion regarding application of 'model rules' regarding maintenance and publishing of Minutes.
2.4.3 Motion regarding increase in subscription fees.
2.4.4 Motion for additional membership category for retired members.
2.4.5 Nominations for a new committee member.
2.5 Strategies for increasing membership were further discussed and progressed.

3 Annual Scientific Meeting
3.1 Planning for the ASM was discussed and progressed.
3.2 The Committee agreed that attendance at conference functions should be for conference participants only.
3.3 Dr Davis was unavailable to advise on any progress in planning for the 2007 meeting.
3.4 Options for a theme for the 2008 ASM were discussed. The proposal remains for this meeting to be held in PNG. Further discussion will be held at the ASM committee meeting.

4 Treasurer's report
4.1 The Treasurer reported in writing on the status of the current accounts. The Society is in a positive financial position. General account $43,145; ASM account $5,676; Investment account $96,307.

5 Journal report
5.1 The Editor was unavailable for this meeting. A comprehensive journal report will be provided at the AGM.
5.2 Dr Acott requested the committee members be prepared to discuss the proposed amalgamation of the EUBS and SPUMS journals at the next meeting of the Committee at the ASM.

6 Education Officer's report
6.1 In February a new DipDHM was awarded to Dr Glen Hawkins at Prince of Wales Hospital, Sydney.

7 Other business
7.1 SPUMS representative on the ANZCA SIG. Further discussion deferred to the next meeting.
7.2 RACP CIG in Diving Medicine has been established. It needs a SPUMS representative and is seeking a volunteer. Current chair is Professor Des Gorman. To be further discussed at the next meeting.
7.3 Complaint of misconduct by a SPUMS member performing dive medicals. Dr Acott has responded to the Victorian Medical Board request with advice regarding the recommended elements of the SPUMS Diving Medical Examination.

8 The next meeting is proposed for Sunday 4 June 2006 (lunchtime) in Fiji at the ASM.

Closed: 1005 hr

Minutes of the Annual General Meeting of SPUMS held at Pearl South Pacific Resort, Pacific Harbour, Fiji, on Wednesday 7 June 2006

Opened: 1715 hr

Present: All members attending the Annual Scientific Meeting

Apologies: S Sharkey, D Vote, G McGeoch, G Hawkins, M Walker, R Walsh

1 Minutes of the 2005 AGM
Minutes of the 2005 AGM were circulated at the meeting and had been previously published in the *SPUMS Journal* 2005; 35: 97.
Motion that the minutes be taken as read and is an accurate record.
Proposed Dr R Walker, seconded Dr C Lee, carried.

2 Matters arising from the minutes
Nil

3 Annual reports
3.1 President's report
3.2 Secretary's report (read by Dr R Walker in Dr S Sharkey's absence)
3.3 Education Officer's report

4 Annual financial statement and Treasurer's report
Presented by Dr G Williams.
Motion that the financial statements be accepted.
Proposed Dr C Acott, seconded Dr V Haller, carried.

5 Subscription fees for the coming year
The previous Treasurer, Dr A Patterson proposed that there be an increase in the annual subscription fee to $A130.00 plus GST for full members and $A70.00 plus GST for associate members.
Proposed Dr G Williams, seconded Dr C Acott, carried.

6 Election of office bearers
The following were elected/appointed:
Education Officer: Dr F Sharp (appointed)
Committee Member: Dr G Hawkins (elected)
Treasurer: Dr G Williams (elected)
Public Officer: Dr V Haller (appointed)

7 Business of which notice has been given
7.1 Motion re amendments to the Constitution required by Consumer Affairs
Proposed Dr G Williams, seconded Dr C Acott, carried.
7.2 Motion re adoption of rules for publishing of minutes
Proposed Dr R Walker, seconded Dr C Acott, carried.
7.3 Motion re adoption of additional membership category for retired members

Proposed Dr R Walker, seconded Dr C Acott, carried.
7.4 Motion to make Martin Sayer a full member of the Society
Proposed Dr M Davis, seconded Dr C Acott, carried.

Closed: 1800 hr

President's report 2006

The Society has grown considerably since I was President in 1987. The recent Committee has many new faces on it. Fortunately the 'changing of the guard' has been smooth; the future of SPUMS is in their hands. Following in the 'large footsteps' of Robyn has been made easier by her continued input and support.

It is a pleasure to work with Sarah Sharkey, the SPUMS Secretary, I thank her for her assistance. I would like to acknowledge the work Cathy Meehan did as Secretary and take this opportunity to thank her for her 12-year input into the Society. I would like to thank our 'Mr Fixit', Steve Goble and other members of the Committee (David Vote, Christine Lee and David Smart) for their help throughout the year.

I acknowledge the excellent work of Associate Professor Michael Davis, the Editor of the *SPUMS Journal*. The Journal is the flagship of the Society and the success of the Society is closely related to its continued publication. It is unfortunate that members still publish original work in other journals and not the Society's despite the *SPUMS Journal* having EMBASE recognition.

Unfortunately my presidency started with the resignation of the Treasurer, Dr A Patterson, who brought a high degree of financial accountability to the Society. The Treasurer's job seems to be a poisoned chalice. Dr G Williams has volunteered to become the current Treasurer and I know he will continue the excellent financial management started by Dr Patterson. I thank Dr Patterson for his excellent work.

My vision for SPUMS:

1 To aid Mike in building a better journal. John Knight started the Journal's academic improvement and Mike has continued this trend. Amalgamation with the European Undersea Barometric Society's journal is presently being considered by the Committee.
2 I have instituted a three-year plan for the Annual Scientific Meeting so that we can plan more effectively and change the format of the conference if necessary.
3 To increase our membership.

Finally I would like to thank Robyn for her efforts in setting up the new SPUMS website. The future development of SPUMS will depend on this website. Thank you all for coming.

Chris Acott

Secretary's report 2006

This last year has sadly seen the end of a 12-year tenure by Cathy Meehan as Secretary of SPUMS. Cathy's loyalty, commitment and endurance in the position were remarkable and appreciated. Twelve years of corporate knowledge and experience are not easy to follow. In addition to a new Secretary the Committee welcomed a new (but affectionately recycled) President and two new committee members. More recently the Treasurer's position became vacant and Dr Guy Williams kindly volunteered to step into it. Since the 2005 AGM the Committee has formally met four times (including two teleconferences and two face-to-face meetings) and has been progressing a number of issues – some of which are only briefly summarised here.

SPUMS membership: Current membership is 867 members including 112 outstanding renewal payments. The exact number of new members since the last AGM is not known at this stage but only 6 resignations have occurred. A number of initiatives to improve the current SPUMS membership are being explored by the Committee. Among these is the introduction of the new SPUMS website. Since going live the number of membership applications has risen noticeably. Other initiatives that are being considered include changes to membership categories, more aggressive marketing and recruitment, and SPUMS journal amalgamation.

SPUMS website: The new SPUMS website went live in October 2005. As reflected in the membership applications it is anticipated to play an important role in improving the profile of the Society. Improvements continue to be made to the resources available on it and feedback from members and visitors is an important part of its development. Many thanks go to Robyn Walker for her tireless efforts in setting it up.

One of our main focuses in the coming year will continue to be developing initiatives to improve membership numbers within the Society and as always your input into this is valued.

Sarah Sharkey

Education Officer's report 2006

This is my first and last report as Education Officer. The Education Officer is appointed by the Committee and Dr Fiona Sharp has agreed to take over the role. I will continue to help and will continue on the Academic Board. I wish Fiona all the best, I am sure she will do a great job.

Five candidates have been awarded the Diploma in the past two years. The standard has been high. Four candidates at present have shown interest but have yet to submit their final submission.

Chris Acott

Treasurer's report 2006

I have taken over as Treasurer from the beginning of the current SPUMS financial year. The current (31 May 2006) state of our finances is:

- General account $34,205
- ASM account $7,615
- Reserve account $97,375

Attached are the audited accounts for the year ending 31 December 2005.

In 2006 I will be reviewing SPUMS' banking arrangements in order to streamline payments to our creditors. Current arrangements require dual signatures on all financial transactions; currently few (one) banks offer dual-authorisation online banking. These options will be pursued with the view to reduce costs and improve efficiency.

The new SPUMS website should enable membership transactions to be conducted online with resultant reduction in costs and improved efficiency. The online system may then flow on to ASM registrations and others items of general revenue (such as Journal CD purchases).

Guy Williams

Audit report to the members of the South Pacific Underwater Medicine Society

I have conducted various tests and checks as I believe are necessary considering the size and nature of the Society and having so examined the books and records of The South Pacific Underwater Medicine Society for the year ended 31 December 2005 report that the accompanying Income and Expenditure and Balance Sheet have been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the year then ended.

Dated: 1 June 2006

David S Porter, Chartered Accountant
Mona Vale, New South Wales 2103

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY STATEMENT OF INCOME AND EXPENDITURE FOR THE YEAR ENDED 31 DECEMBER 2005

	2005	2004
INCOME		
Subscriptions and registrations	97,835	91,799
Interest	4,648	4,273
Advertising and Journal sales	-	323
ASM 2004	2,532	37,370
ASM 2005	31,719	3,100
Sundry income	2,221	807
	138,955	**137,672**
EXPENSES		
Amortization of website	10,307	6,667
ASM costs	41,128	36,110
ASM registrations previous year	-	10,333
Bad debt written off	-	5,889
Secretarial wages	13,978	13,868
Stationery, printing, postage	2,548	1,332
Journal	20,354	37,907
Committee expenses	9,149	5,509
Computer equipment	491	2,778
Mail forwarding	600	456
Miscellaneous/Subscriptions	309	231
Bank charges and card charges	5,196	4,415
Audit	2,100	2,000
Editors honorarium and expenses	21,394	17,350
Insurance	5,727	5,961
	133,281	**150,806**
SURPLUS/(DEFICIENCY) FOR YEAR	**$5,674**	**$(13,134)**

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Mona Vale, NSW 2103.
Dated: 1 June 2006

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY BALANCE SHEET AS AT 31 DECEMBER 2005

	2005	2004
MEMBERS' FUNDS		
Balance at 1 January 2005	124,487	132,237
Funds provided by NZ Chapter	-	4,384
Surplus/(Deficiency) for year	5,674	(13,134)
	$130,161	**$124,487**
represented by:		
NON-CURRENT ASSETS		
SPUMS website, at cost	30,620	20,000
Less, provision for amortization	(16,974)	(6,667)
	13,646	**13,333**
CURRENT ASSETS		
ANZ Access Cheque Account	7,162	46
ANZ VZ Plus	95,165	100,590
ANZ SPUMS Annual Scientific Meeting	3,853	6,949
BNZ Achiever Savings	2,812	2,307
2006 ASM income less expenses in advance	6,403	-
GST recoverable	1,120	1,262
	116,515	111,154
NET ASSETS	**$130,161**	**$124,487**

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Mona Vale, NSW 2103
Dated: 1 June 2006

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY MOVEMENTS ON BANK BALANCES FOR THE YEAR ENDED 31 DECEMBER 2005

		2005	2004
OPENING BALANCES			
ANZ bank	- ASM account	-	6,447
	- Access cheque account	46	13,632
	- SPUMS Annual Scientific Meeting	6,949	-
	- BNZ Achiever Savings	2,307	-
	- VZ Plus	100,590	96,400
		109,892	116,479
add, **RECEIPTS**		142,967	137,666
		253,057	254,145
less, **PAYMENTS**		142,967	144,253
CLOSING BALANCES			
ANZ bank	- Access cheque account	7,162	46
	- VZ Plus	95,165	100,590
	- SPUMS Annual Scientific Meeting	3,853	6,949
BNZ Achiever Savings		2,812	2,307
		$109,470	**$109,892**

NOTE: Receipts and Payments above include some 2006 year receipts and payments and may include Balance Sheet items which are not included in the Income and Expenditure statement.

Letter to the Editor

The future of Project Stickybeak

Dear Editor,

It is with some frustration that I read Douglas Walker's letter in the Journal about the future of Project Stickybeak and his apparent lack of success in finding a 'successor'.[1]

I want to make it absolutely clear, as I have attempted to do so with Douglas on many occasions, that DAN Asia-Pacific fully intends to collect and report on dive fatalities as an ongoing project. We have been gearing up to this and, in fact, have relatively recently advertised a part-time research position incorporating this role, among others. It is our intention to publish reports from 2003 onwards.

We intend to work either with or without Douglas to expand dive fatality data collection and reporting throughout the Asia-Pacific. Douglas should feel comfortable that his great legacy in this important area will be continued and expanded.

Reference

1 Walker D. A successor for Project Stickybeak. *Diving and Hyperbaric Medicine*. 2006; 36: 110.

John Lippmann
Executive Director,
Divers Alert Network (DAN) Asia-Pacific,
Box 384, Ashburton, VIC 3147, Australia
E-mail: *<www.danasiapacific.org>*

Key words
Letters (to the Editor), diving accidents, diving deaths, research

Editor's comment: It is vital that DAN Asia-Pacific ensures there is active medical supervision and participation in the review process by an experienced diving physician.

First graduate of The University of Auckland's postgraduate diploma in diving and hyperbaric medicine

Dr Alexander ('Sandy') Inglis, an emergency medicine specialist and hyperbaric consultant at Christchurch Hospital, New Zealand, has been awarded a Postgraduate Diploma in Medical Science – Diving and Hyperbaric Medicine.

Sandy is the first graduate of this international distance-learning programme established in late 2004. Currently there are 16 physicians undertaking the programme under the direction of Associate Professor Michael Davis.

Book reviews

Report on decompression illness, diving fatalities and Project Dive Exploration

DAN's annual review of recreational scuba diving injuries and fatalities, 2004 and 2005 editions (based on 2002 and 2003 data)

150 and 140 pages respectively, soft cover
ISBN (2004): 0-9673066-5-5
ISBN (2005): 0-9673066-7-1
Durham, NC: Divers Alert Network
6 West Colony Place, Durham, NC 27705, USA
Phone: (+1)-919-684-2948
Fax: (+1)-919-490-6630
E-mail: <dan@DiversAlertNetwork.org>
Copies can be ordered online from <www.DiversAlertNetwork.org>
Price: 2004 edition US$7.00 (2005 price unavailable)

The 2003 edition of this publication was reviewed previously.[1,2] The annual review of DAN data on recreational scuba injuries and fatalities includes data from Project Dive Exploration (PDE). It collects prospective and accurate data on diving habits from selected populations of divers. The 2004 and 2005 editions follow identical formats except the 2004 edition includes a chapter on five-year trends (1998–2002) in diving activity for PDE, injuries and fatalities compared with 1987 to 1997. This review briefly summarises these trend data and some of the interesting features of the annual reviews.

PDE data for 1998 to 2002 show no trends for age of divers, sex, years since training, days in dive series or dives per day. Average maximum depth was 26 to 30 metres' sea water (msw) without trend, and women had a mean of 1.5 m less than men. Nitrox diving increased during the period. Reported buoyancy problems decreased during the period while the incidence of rapid and out-of-air ascents was 10 times higher in injured divers.

Dive injuries in the same period decreased in the USA but increased in Central America and the Caribbean. The mean age of injured divers increased from 37 to 39 years. This follows an increase between 1987 and 1997 from 33 to 37. Injured divers over 50 increased from 7% to 15%. This may indicate an ageing diving population despite the lack of such a trend in the PDE data. The number of injured divers using computers increased from 20% in 1987 to 60% in 1998 and 70% in 2002. In spite of this, very few depth-time profiles were available from the computers. The median days of diving and the number of dives in a dive series before injury increased during the period. Women made fewer dives over more days than men prior to injury. Mean depth of injury increased by 3 msw from the previous period and men were injured 3 msw deeper than women on average. The percentage of injured divers using nitrox increased from 6% to 12% during the period, reflecting the increase in nitrox diving.

The delay to recompression remained fairly constant at a median of one day although the mean varied widely indicating some very long delays. Median recompression treatments increased from one to two and mean from two to three during the period, although overall they were similar to the previous period. Complete relief of symptoms at discharge increased to 70% after showing a gradual increase from 50% in 1989, suggesting the efficacy of recompression therapy may have improved.

The publications review 89 diving fatalities in each of 2002 and 2003. This is not a complete record of US recreational diving deaths but represents a large recent series and thus is of interest. The cause of death in the majority was given as drowning. The DAN data allow comparison of different diver populations, making it possible to draw some fascinating inferences from the data.

There were no teenage fatalities in 2002 and only one 18-year-old in 2003, which is fewer than previous years. The median age continues to be in the mid 40s with males a little older than females and divers who died five to six years older than injured divers or PDE divers. Less than a quarter were of normal body weight compared with over half of the other groups. This information may reinforce the need for medical review of overweight divers, especially those aged over 40. One third had been certified for over 10 years and half were doing their first dive for over one year. However, divers who died had a lower average number of years since certification than injured divers or PDE divers, so experience may have some advantage even if age does not! Trainees and technical divers seemed to die more frequently than their representation in other diving populations. Of similar interest is the lower incidence of previous medical problems in fatalities, except for hypertension and heart disease which were higher. Fatalities had a mean maximum depth of 20 msw compared with injuries, 30 msw, and PDE, 27 msw. In this series, and not unexpectedly, running out of air, buoyancy problems and rapid ascent all appear to represent a significant risk of death, and rapid ascent a particular risk of injury.

The 2005 report includes a new chapter on breath-hold diving incidents. DAN has collected data on 145 fatal cases since 1994 and these data to 2004 are reported here. This is a useful addition to the report, reflecting the increase in deep freediving as a sport and in offshoots such as extreme blue-water spear fishing. Eighty-eight per cent were male with an average age of 38 years. The cause of death was usually drowning with contributing factors frequently not identified clearly. The most common factor was entanglement with kelp and lines. This is recognisable after the event unlike the lack of features post mortem of shallow water blackout or other medical causes. Thus, medical factors in diving deaths are frequently not identified. Four cases involved boat strikes and two shark attacks. Solo breath-hold diving featured in some fatalities and careful dedicated support is clearly important for more extreme dives. Alcohol featured in at least two cases. Much of this chapter is a discussion of the risks of breath-hold diving and the data are much less detailed.

The publication ends with case reports of dive injuries, deaths and breath-hold incidents. These add a personal element to the preceding dry data and although written in a medical, dispassionate style, resonate poignantly with anyone who enjoys diving and anecdotes.

References

1 DAN. *Report on decompression illness, diving fatalities and Project Dive Exploration. The DAN annual review of recreational scuba diving injuries and fatalities, based on 2001 data*. Durham, NC: Divers Alert Network; 2003. *SPUMS J*. 2004; 34: 111-2.
2 DAN International. Project Dive Exploration 2003. *SPUMS J*. 2004; 34: 222-3.

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Key words
Book review, accidents, incidents, deaths, decompression illness, recreational diving, DAN – Divers Alert Network

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Assessment of diving fitness for scuba divers and instructors

Peter Bennett, Frans Cronjé and Ernest Campbell

241 pages, hard cover
ISBN 1-930536-31-3
Flagstaff, Arizona: Best Publishing Company; 2006
PO Box 30100
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E-mail: <divebooks@bestpub.com>
Copies can be ordered online from <www.bestpub.com>
Price: US$29.95 + P&P

From abdominal cramps to vomiting, this book provides advice in relatively simple, non-medical language to help divers understand the impact of medical conditions on diver safety. Part one sets the scene by explaining the important role that the instructor can play in evaluating diving fitness; part two highlights the responsibilities of the diver; and part three provides factual information on common and important medical conditions and disorders that have an impact on diving.

The first part of the book helps instructors to consider physical, physiological and psychological factors in the risk-analysis assessment of divers in the context of the demands of the diving environment. The authors point out that nothing can replace the face-to-face discussions between a diver and a physician trained in diving medicine. However, they believe the book will empower the instructor to determine when input from a diving physician is mandatory, or strongly recommended. The book also advises the instructor on how to avoid pitfalls in diving fitness, how to sensibly screen prospective divers or dive students for medical problems, and what to do if a problem is discovered.

Part two conveys to the diver the message that 'fitness' for diving is not static and that health is an ever-changing, dynamic phenomenon. Importance is placed on maintaining lifelong health and fitness in an attempt to prevent the conditions associated with the ageing process. There is also a very useful section on 'diving nuisances', helping the diver to understand common problems such as ear problems, headaches and motion sickness, and giving practical advice on prevention and treatment, including suitable medication options.

Part three is divided into body systems and makes up the largest section of the book, with 125 pages covering the A to Z of common and important medical conditions and disorders with an impact on diving. Finally, there are nearly seventy scientific references in a good bibliography and a useful list of medical acronyms and abbreviations.

I have only two minor criticisms of the book. Firstly, the units used to express laboratory results, such as lipid levels, are given in mg^{-1}, which may not mean much to readers who live in countries where laboratory test results are expressed in $mmol/l^{-1}$. A simple equivalent in parenthesis would have been a useful addition to help non-American readers relate to what is a high or low value. Secondly, because the references are listed alphabetically, rather than cited throughout the main text or at the end of each chapter, it is less easy to identify relevant references for further reading on a particular topic of interest.

In summary, this book provides for divers and diving instructors a refreshing, balanced and factual explanation of the risks of diving with medical conditions. Only a few medical disorders are regarded as absolute contra-indications to diving. For most conditions the authors present a risk analysis and recognise that, ultimately, it will be the divers themselves who take responsibility for the decision to dive or not.

These authors are all well-respected authorities in diving medicine and they have to be congratulated on putting together a comprehensive reference book of medical fitness, in a language that non-medical people will understand. This 241-page hardback book is a must read for every instructor and should have a place in every dive operator's reference library.

Lynn Taylor
President, New Zealand Underwater
PADI Instructor

Key words
Book review, fitness to dive, medical conditions and problems, recreational divers, scuba diving

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The poetry doctor

A fishy dilemma

They come regularly from outer space
Crash landing in a bubbling, frothing ball,
Seemingly lost and out of control,
Fins thrashing wildly to stabilise their long entry fall.

They hover, peering downwards through a never-blinking eye,
Groping at loose flaps of skin and banded warty waist
Till all seems settled when they quickly sink
Leaving an umbilical bubbling wake in their downward haste.

How ugly they are with their long gangly fins,
Bubbles belching from a knobbly beak.
Most are black and drab, bleak as funeral dress
Whilst some sport a vivid red or yellow streak.

How slow and clumsily they crash around
Stirring up the sediment and sand.
Some are photoluminescent and flash
For reasons difficult to understand.

They are so unadventurous and timid
Too scared to go into the dark or deep
Moving around in small shoals
Yet following a leader like sheep.

They are quickly bored and depart
Slowly surfacing, readying to fly
And return to their earthy world
Through the surging stargate sky.

Why do they keep coming?
What is the attracting mystique?
What are they thinking? What are they escaping?
What really do they seek?

John Parker
<www.thepoetrydoctor.com>

THE UNIVERSITY OF AUCKLAND
FACULTY OF MEDICAL AND HEALTH SCIENCES

Master of Medical Science
Postgraduate Diploma in Medical Science
Diving and Hyperbaric Medicine

Enquiries from registered medical practitioners are now being accepted for 2007 for the Masters and Postgraduate Diploma programmes in diving and hyperbaric medicine.

Faculty: Des Gorman, Michael Davis (Course Director), Simon Mitchell, Chris Acott, Kathleen Callaghan, William Baber and Drew Richardson.

Overview: These are distance-learning programmes, available internationally without a resident component in Auckland. However, attendance at a recognised short course in diving medicine (such as those listed here) is a prerequisite. Graduates will be able to practise effective clinical diving medicine in a primary care setting or to embark on clinical practice within a hyperbaric medicine environment.

For further information, including fees, please contact the Course Coordinator:
Upendra Wickramarachchi
Phone: +64-(0)9-373-7599, extn 83058
Fax: +64-(0)9-373-7006
E-mail: <u.wicks@auckland.ac.nz>

Or submit an online Expression of Interest in this subject at the website: **<www.health.auckland.ac.nz>**
by clicking on the Quicklink to Postgraduate Study

2007 ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS' UNDERWATER MEDICINE COURSE

Dates: 09 to 20 July 2007
Venue: HMAS Penguin, Sydney
Cost: $1833.00 (tbc)

The Medical Officers' Underwater Medicine Course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Considerable emphasis is placed on the contra-indications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving-related illnesses.

For information and application forms contact:
The Officer in Charge, Submarine & Underwater Medicine Unit, HMAS PENGUIN,
Middle Head Road, Mosman, 2088 NSW, Australia
Phone: +61-(0)2-9960-0572
Fax: +61-(0)2-9960-4435
E-mail: <Sarah.Sharkey@defence.gov.au>

SPUMS

ANZ HYPERBARIC MEDICINE GROUP of SPUMS

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Contact for information:
Ms Gabrielle Janik, Course Administrator
Phone: +61-(0)2-9382-3880
E-mail: <Gabrielle.Janik@sesiahs.health.nsw.gov.au>

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DMT Refresher Course

December 2006 04/12/06 to 08/12/06

For further information or to enrol contact:
The Director, Hyperbaric Medicine Unit
Royal Adelaide Hospital, North Terrace
South Australia 5000 or
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lmirabel@mail.rah.sa.gov.au>

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For further information, please visit our website <www.eubs2007.org> or contact:
Dr Adel Taher, Secretary General of 33rd EUBS Annual Scientific Meeting
E-mail: <info@eubs2007.org>
Mobile: +20 12 212 4292 (24 hours)

BRITISH HYPERBARIC ASSOCIATION ANNUAL MEETING 2007

Dates: 01 to 04 November 2007 (pre-meeting diving programme 29 October to 01 November)
Venue: Oban, Scotland

For information contact: BHA 2007, Dunstaffnage Hyperbaric Unit, Scottish Association for Marine Science, Oban, Argyll, Scotland PA37 1QA
E-mail: <info@bha2007.org>
Website: <www.bha2007.org>

ASIAN HYPERBARIC & DIVING MEDICAL ASSOCIATION (AHDMA)
THIRD SCIENTIFIC MEETING 2007

Dates: 12 to 15 April 2007
Venue: Sanur Paradise Plaza Hotel, Bali, Indonesia

Registration details and further information contact:
E-mail: <secretary@ahdma.com>
Website: <www.ahdma.com>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA
All enquiries to:
Diving Historical Society Australia, SE Asia,
PO Box 2064,
Normansville, SA 5204,
Australia
Phone: +61-(0)8-8558-2970
Fax: +61-(0)8-8558-3490
E-mail: <bob@hyperbarichealth.com>

UNDERSEA and HYPERBARIC MEDICAL SOCIETY
Annual Scientific Meeting 2007
Preliminary Announcement

Dates: 14 to 16 June 2007
Venue: The Ritz-Carlton, Kapalua, Maui

General information and online registration can be found at <http://www.uhms.org/Meetings/AMMeetingsMain.htm>

For additional information:
Lisa Wasdin, c/o Undersea and Hyperbaric Medical Soc.
PO Box 1020, Dunkirk, Maryland 20754, USA
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E-mail: <lisa@uhms.org>

INTERNATIONAL CONGRESS ON DIVING & HYPERBARIC MEDICINE, MUSCAT 2006
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Dates: 2 to 7 December 2006
Venue: Muscat, Oman

International Faculty includes Professors David Elliott, Des Gorman and Richard Moon

The congress is accredited by the University of Auckland for its programmes in diving and hyperbaric medicine.

For additional information:
Sultanate of Oman, Said Bin Sultan Naval Base, Medical Center, Royal Navy of Oman
PO Box: 839, Postal Code: 111 Muscat
Phone: +968-26-346832
Fax: +968-26-346367
E -mail: <drhassan@dhm.org.om>
Website: <www.dhm.org.om>

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Guest speakers Associate Professor Mike Davis, Professor Des Gorman, and Mr Dick Clarke

For further information contact: Czes Mucha
E-mail: <cmucha@mail.rah.sa.gov.au>
Phone: +61-(0)8-8222-5121
Fax: +61-(0)8-8232-4207

Instructions to authors

(revised June 2005)

Diving and Hyperbaric Medicine welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts, including SPUMS Diploma theses, will be subject to peer review. Accepted contributions will be subject to editing.

Contributions should be sent to:
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C/o Hyperbaric Medicine Unit, Christchurch Hospital,
Private Bag 4710, Christchurch, New Zealand.
E-mail: <spumsj@cdhb.govt.nz>

Requirements for manuscripts

Documents should be submitted electronically on disk or as attachments to e-mail. The preferred format is Word 97 for Windows. Paper submissions will also be accepted. All articles should include a **title page**, giving the title of the paper and the full names and qualifications of the authors, and the positions they held when doing the work being reported. Identify one author as correspondent, with their full postal address, telephone and fax numbers, and e-mail address supplied. The text should be subdivided into the following sections: an **Abstract** of no more than 250 words, **Introduction**, **Methods**, **Results**, **Discussion**, **Acknowledgements** and **References**. Acknowledgments should be brief. References should be in the format shown below. Legends for tables and figures should appear at the end of the text file after the references.

The text should be double-spaced, using both upper and lower case. Headings should conform to the current format in *Diving and Hyperbaric Medicine*. All pages should be numbered. Underlining should not be used. Measurements are to be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included.

The preferred length for original articles is 3,000 words or less. Inclusion of more than five authors requires justification as does more than 30 references per major article. Case reports should not exceed 1,500 words, with a maximum of 10 references. Abstracts are also required for all case reports and review papers. Letters to the Editor should not exceed 500 words (including references, which should be limited to five per letter). Legends for figures and tables should generally be less than 40 words in length.

Illustrations, figures and tables should not be embedded in the wordprocessor document, only their position indicated. No captions or symbol definitions should appear in the body of the table or image.

Tables are to be in Word for Windows, tab-separated text rather than using the columns/tables option or other software and each saved as a separate file. They should be double-spaced and each in a separate file. No vertical or horizontal borders are to be used.

Illustrations and figures should be in separate files in TIFF or BMP format. Our firewall has a maximum size of 5 Mb for incoming files or messages with attachments.

Photographs should be glossy, black-and-white or colour. Posting high-quality hard copies of all illustrations is a sensible back-up for electronic files. Colour is available only when it is essential and may be at the authors' expense. Indicate magnification for photomicrographs.

Abbreviations may be used once they have been shown in brackets after the complete expression, e.g., decompression illness (DCI) can thereafter be referred to as DCI.

References

The Journal reference style is the 'Vancouver' style (*Uniform requirements for manuscripts submitted to biomedical journals*, updated July 2003. Web site for details: <http://www.icmje.org/index.html>). In this system references appear in the text as superscript numbers at the end of the sentence and after the full stop.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). Examples are given below:

1 Freeman P, Edmonds C. Inner ear barotrauma. *Arch Otolaryngol.* 1972; 95: 556-63.
2 Hunter SE, Farmer JC. Ear and sinus problems in diving. In: Bove AA, editor. *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Saunders; 2003. p. 431-59.

There should be a space after the semi-colon and after the colon, and a full stop after the journal and the page numbers. Titles of quoted books and journals should be in italics. Accuracy of the references is the responsibility of authors.

Any manuscript not complying with these requirements will be returned to the author before it will be considered for publication in *Diving and Hyperbaric Medicine*.

Consent

Studies on human subjects must comply with the Helsinki Declaration of 1975 and those using animals must comply with National Health and Medical Research Council Guidelines or their equivalent. A statement affirming Ethics Committee (Institutional Review Board) approval should be included in the text. A copy of that approval should be available if requested.

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+64-9-445-8454 (International)
The toll-free number 0800-4-DES111 can only be used in New Zealand

The DES numbers are generously supported by DAN-SEAP

PROJECT STICKYBEAK

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

Information may be sent (in confidence) to:
Dr D Walker
PO Box 120, Narrabeen, NSW 2101, Australia.
Enquiries to: <diverhealth@hotmail.com>

DIVING INCIDENT MONITORING STUDY (DIMS)

DIMS is an ongoing study of diving incidents. An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report anonymously any incident occurring in your dive party. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver injury. Using this information to alter diver behaviour will make diving safer.

Diving Incident Report Forms (Recreational or Cave and Technical)
can be downloaded from the DAN-SEAP website: <www.danseap.org>
They should be returned to:
DIMS, 30 Park Ave, Rosslyn Park, South Australia 5072, Australia.

DIVING-RELATED FATALITIES RESOURCE

The coronial documents relating to diving fatalities in Australian waters up to and including 1998 have been deposited by Dr Douglas Walker for safe keeping in the National Library of Australia, Canberra. Accession number for the collection is: MS ACC 03/38.

These documents have been the basis for the series of reports previously printed in this Journal as Project Stickybeak. They are available free of charge to *bona fide* researchers attending the library in person, subject to an agreement regarding anonymity.

It is hoped that other researchers will similarly securely deposit documents relating to diving incidents when they have no further immediate need of them. Such documents can contain data of great value for subsequent research.

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