

Diving and Hyperbaric Medicine

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and the European Underwater and Baromedical Society*

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EUBS



Scientific diving in the Canadian high Arctic

Changes in blood glucose during HBOT no different to room air

Is finger-prick blood glucose reliable during HBOT?

DCI or acute coronary syndrome – a diagnostic dilemma

Diving-related deaths in Australian waters in 2009

Pulmonary barotrauma in breath-hold divers

HBOT good for muscle injury in a rat model

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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 and to convene members of each Society annually at a scientific conference

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The Editor's offering

The Tricontinental conference, Réunion2013 was a great success. Physicians and scientists from the Antipodes to almost the Arctic Circle heard and read a diverse range of presentations of a high standard and had a wonderful opportunity to network with colleagues. For me, it was very special to meet for the first time many authors with whom I have corresponded over the years as your Editor. The organisation was first class, especially given some challenging logistics. I would particularly like to record my thanks to the Scott Haldane Foundation and its incredibly hard-working staff, JJ Brandt Corstius and Pascale Kragt-Trompetter. Their contributions were vital to the success of the meeting. I believe that all three participating societies will be the stronger, and the links forged will enhance this journal into the future. Here's to the next joint meeting!

EUBS and SPUMS joined forces for *Diving and Hyperbaric Medicine* following a meeting that Robyn Walker and I had with the EUBS Executive at the 2007 ASM at Sharm el Sheikh and an informal gathering that evening of would-be editorial board (EB) members. Since then, almost all the business of the EB has been conducted electronically. The Réunion meeting provided an opportunity for the first formal face-to-face meeting of the Board, of whom nine members were able to attend, some wearing two hats – as EB members and as members of the executives of our two societies. This allowed us to address both governance and academic issues at the same time in a very full agenda.

Amongst the many items discussed was the embargoing of published articles. There are increasing demands for free access, especially for publically funded research. This does not sit well with small specialty journals funded by societies. Current policy of DHM is for articles to be embargoed for one year following publication and then to be placed in the Rubicon Foundation database. In reality, Rubicon, which is funded largely by donation, is behind in placing DHM (and back issues of *SPUMS Journal*) on the database. The EB invited both executives to consider donating specifically toward bringing this fully up to date and we invite society members to support the Foundation as well. Some authors are now asking for their papers to be unembargoed immediately, and the executives will need to look into a fee structure for this, as it impinges in a negative way on the financial foundations of the Journal if everything is freely available on the web.

There was general agreement that we need to move to electronic publishing in the relatively near future. Whether this is in parallel with a print copy remains to be seen at this stage. However, the editorial office will be converting its management of papers, from submission to publication, to a fully electronic process in 2015, and it follows naturally to provide a journal that can be readily viewed on your

computer, smart phone, iPad, smart TV, etc. The costs of producing a print journal are steadily increasing at the same time as subscriber numbers overall remain constant. Experience of other small journals such as ours is that there are substantial savings to be made by converting to a fully electronic journal. For instance, a journal in New Zealand saved a six-figure sum when it moved from print to electronic publication, with no loss of subscriptions; indeed, subscriptions actually rose.

We reviewed the “*Instructions to Authors*”, a revision of which will appear in 2014 along with additional advice regarding authorship and English as a second language. Peer review for the Journal appears to be generally working well, with many authors commenting that they felt going through this process had helped them considerably to improve their papers. Regrettably some papers are rejected, sometimes after a great deal of time and effort from reviewers and myself. We have a widening resource of external reviewers to help the EB; new volunteers reveal themselves, whilst we drop off the other end reviewers who consistently do not meet deadlines. A deficiency in biostatistical review was acknowledged. Though many EB members have some training in statistics, it was recognised that we need to improve support in this area to meet ever-changing, increasingly sophisticated, modern standards. Knowing how to do a chi-square test or Student's *t*-test is no longer the name of the game! Since the meeting, we have found just the resources we need: one in Scotland, Tom Wilding at SAMS in Oban, and the other, Chris Frampton here at the University of Otago, Christchurch – as far from each other as it is geographically possible! Many on the EB are very busy people and sometimes cannot meet their journal commitments for periods, so more hands on deck are needed. To this end, I am delighted to welcome three new members – Professor Claus-Martin Muth, Ulm, Germany, Associate Professor Monica Rocco, Rome, Italy and Professor Erika Schagatay, Ostersund, Sweden.

Finally, it is with regret that we learn that Peter Müller is retiring next year as European Editor. Peter was the key person in establishing the *European Journal of Underwater and Hyperbaric Medicine* for EUBS and then in negotiating the merger of that journal with the *SPUMS Journal*. EUBS has a great deal to thank Peter for and I trust he will have a fitting sendoff in Wiesbaden in September! I have enjoyed immensely working with him but look forward with some excitement to learning who will be my next colleague in this role.

Michael Davis

Prof. Frithjof Küpper, Aberdeen University, collecting the Arctic seaweeds *Saccharina latissima* and *Laminaria solidungula* in the Canadian high Arctic. Photo courtesy Hugh Brown, UK National Facility for Scientific Diving

Original articles

Provisional report on diving-related fatalities in Australian waters 2009

John Lippmann, Christopher Lawrence, Andrew Fock, Thomas Wodak and Scott Jamieson

Abstract

(Lippmann J, Lawrence CL, Fock A, Wodak T, Jamieson S. Provisional report on diving-related fatalities in Australian waters 2009. *Diving and Hyperbaric Medicine*. 2013 December;43(4):194-217.)

Introduction: An individual case review of diving-related deaths reported as occurring in Australia in 2009 was conducted as part of the DAN Asia-Pacific Dive Fatality Reporting Project.

Method: The case studies were compiled using reports from witnesses, the police and coroners. In each case, the particular circumstances of the accident and details from the post-mortem examination, where available, are provided.

Results: In total, there were 21 reported fatalities (two more than in 2008), including 18 males and three females. Twelve deaths occurred while snorkelling and/or breath-hold diving, eight while scuba diving and one while using surface-supply breathing apparatus. Apneic hypoxia continues to be a problem with breath-hold divers and appears to have caused the death of three victims in this series. Cardiac-related issues were thought to have been the disabling injury in the deaths of at least three snorkel divers and at least three scuba divers. One of the victims was a student who became separated from her instructor on an introductory scuba dive in poor visibility.

Conclusions: Apneic hypoxia, pre-existing medical conditions, snorkelling or diving alone, separation and inadequate supervision were once again features in several deaths in this series.

Key words

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

Introduction

Each year in Australia there are deaths associated with snorkelling and diving using compressed gas (i.e., scuba or surface-supply breathing apparatus, SSBA). Although some accidents are unavoidable, many might have been avoided through better education about the proposed activity and/or associated risks; appropriate medical screening; greater experience; common sense; improved supervision; or better equipment maintenance and design. The aim of the Divers Alert Network (DAN) Dive Fatality Reporting Project (incorporating Project Stickybeak) is to educate divers and the diving industry and to inform diving physicians on the causes of fatal dive accidents in the hope of reducing the incidence of similar accidents in the future and of detecting, in advance, those who may be at risk. This report includes the diving-related fatalities between 01 January and 31 December 2009 that are recorded on the DAN Asia-Pacific (DAN AP) database. When an accident is unwitnessed, it is often very difficult to determine exactly what has occurred. We have sometimes included considered speculation within the comments to provoke thought about the possible sequence of events.

Methods

As part of its ongoing research into, and reporting of diving fatalities in Australia and elsewhere in the Asia-Pacific region, DAN AP has obtained ethics approval from the Human Research Ethics Committee, Department of Justice, Government of Victoria, Australia to access and

report on data included in the Australian National Coronial Information System (NCIS). In addition, ethics approvals have been sought and obtained from various coronial offices in certain Australian States and Territories. The methodology used for this report was identical to that described previously for the 2004 Australian diving-related fatalities.¹ When considering the possible sequence of events in each case, root cause analysis has been utilised.²

Breath-hold and snorkelling fatalities (Table 1)

BH 09/01

This 63-year-old male was described as a fit and healthy non-smoker and light drinker who attended the gymnasium three times a week. He was a strong swimmer and very experienced snorkeller and spear fisherman. He and his (much younger) buddy went spearfishing at a site that the victim had dived frequently. The conditions were choppy with a slight (0.7 metre) swell and visibility of 5 to 8 metres. The victim was wearing a mask, snorkel, fins, t-shirt and board shorts and carried a speargun.

The pair entered the water into a channel through the rocks. Once clear of this channel, they went in opposite directions and were then out of visual contact for the rest of the dive. After about an hour, when the buddy returned to the entry point, he was unable to see his friend. A short time later, a bystander approached to inform him that his buddy was on the rocks, approximately 100 metres away, apparently dead. That bystander had seen the victim floating face-down and

apparently lifeless near to the rocks. He entered the water, swam to him and, on rolling him over, found the victim to be unconscious with his mask down around his mouth and without a snorkel. He towed him to shore where other bystanders helped to drag the victim onto the rocks. He was found to be unconscious, apneic and without a palpable pulse so bystander Basic Life Support (BLS) was commenced and continued until paramedics arrived and attempted Advanced Life Support (ALS), albeit unsuccessfully.

Autopsy: No measurements were given by the medical examiner (a GP) who described the victim as “*large*”. The examination was limited to the chest. The heart was large, 500 g (normal range (NR)* 365 +/- 71 g), and showed severe coronary atherosclerosis in the proximal left anterior descending (LAD) coronary artery and patchy lesser coronary atherosclerosis generally. The right and left lungs weighed 1,000 g and 800 g respectively (nr 663 +/- 239 g, 583 +/- 216 g) and showed severe pulmonary oedema and free fluid in the major airway, suggesting terminal drowning. The cause of death was given as drowning. Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: Unrecognised ischaemic heart disease even in the apparently fit remains a significant risk factor, especially in the older diver. Had these divers stayed in visual contact and close together during the dive, it is possible that the buddy could have witnessed the incident and might have been able to assist. This may or may not have altered the outcome. Proponents of this ‘same-ocean’ buddy system must understand clearly that they really are on their own if something goes wrong.

Summary: Apparently fit and healthy; strong swimmer and experienced spear fisherman; choppy seas with mild swell; intentional separation from buddy; found unconscious by bystander; BLS unsuccessful; significant coronary vascular disease (CVD) at autopsy; drowning (likely cardiac-related)

BH 09/02

This victim was a 56-year-old male overseas tourist who was visiting the Great Barrier Reef (GBR). He and his wife were partaking in a day trip on a tourist vessel with 17 passengers and two crew. His wife described him as “*a well-rounded athlete*” who “*could swim OK*”, although he rarely entered the water. He had a history of sleep apnea, hypertension (on irbesartan) and an appendectomy three years previously. He was an inexperienced snorkeller but that morning he had snorkelled for about 30 minutes, played beach cricket and hiked up a hill, all prior to lunch. At no stage had he appeared, or complained to his wife of feeling unwell.

* **Footnote 1:** Normal ranges for heart and lung weights expressed by different pathology departments may differ slightly, and so the local ranges, based on age, sex, height and weight, are quoted for each victim.

At least one hour after lunch he went snorkelling with some family members and others from the vessel. He was wearing a mask, snorkel and fins and a full stinger suit. The weather was warm (26°C), there was a moderate wind (10–15 knots), a slight sea, current of about 0.5 knots and the visibility was reported to be good. The group snorkelled at the edge of a drop-off.

About 15 minutes after entering the water, the lookout noticed the victim and some family members snorkelling towards the boat. The victim initially appeared to be swimming well and was not using a ‘noodle’ to provide support. Although his daughter reached the boat and exited the water, the victim appeared to drift past, staring at those on board. The lookout became concerned and shouted “*are you OK?*”, after which the victim raised his arm weakly and appeared to wave goodbye to his wife. The lookout reached the victim just as he began to sink, rolled the victim over and found him to be unconscious. He gave two rescue breaths and towed the victim to the vessel where the skipper helped to drag him aboard and then commenced BLS. Supplemental oxygen was not available. BLS was continued for about 10 minutes as the boat motored to a nearby resort. On arrival, a guest, who identified herself as a nurse, found the victim to be pulseless, with a grey appearance and his eyes rolled back. She reported that she heard the movement of fluid in his stomach. The nurse and skipper continued BLS for several minutes after which resuscitation efforts were abandoned.

Autopsy: The heart weighed 386 g (NR 370 +/- 75 g). There was severe atherosclerotic stenosis of the LAD coronary artery and moderate atheroma of the right coronary artery. There was no scarring of the myocardium. The right and left lungs weighed 832 g and 704 g respectively (NR 651 +/- 241 g, 579 +/- 201 g) and were congested and oedematous. The cause of death was given as myocardial ischaemia. Toxicology: alcohol undetected; irbesartan 0.4 mg kg⁻¹.

Comments: It is possible that this death resulted from a cardiac arrhythmia due to ischaemic heart disease and the triggers present in snorkelling, e.g., immersion and exertion.

Summary: Hypertension and sleep apnea, but apparently healthy; not a strong swimmer; limited snorkelling experience; calm sea with slight current; collapsed while under observation; rapid rescue; BLS unsuccessful; significant CVD at autopsy; likely cardiac death

BH 09/03

The victim, a 26-year-old, male foreign national, was temporarily working in Australia. There is no information on his medical history although, to his friends, he appeared to be fit and healthy. He was reported to be a relatively poor swimmer. Late one afternoon, he and four friends (of the same nationality) went to a beach on the banks of a very large river. Here, the victim and one friend swam before having a snack with others on the riverbank. It was reported that the

Table 1
Summary of snorkelling and breath-hold
BNS – buddy not separated; BSB – buddy separated before problem;

ID	Age (yr)	Gender	Height (m)	Weight (kg)	BMI (kg m ⁻²)	Training	Experience
BH09/01	63	M	n/s	n/s	–	n/s	yes
BH09/02	56	M	182	85	25.7	n/s	some
BH09/03	26	M	n/s	n/s	–	nil	nil
BH09/04	71	M	176	89	28.7	nil	yes
BH09/05	61	F	165	88	32.3	nil	some
BH09/06	76	M	163	61	23.0	nil	nil
BH09/07	31	M	172	66	22.3	trained	yes
BH09/08	28	M	182	74	22.3	nil	nil
BH09/09	31	M	169	88	30.8	trained	yes
BH09/10	20	M	185	95	27.8	n/s	yes
BH09/11	68	M	174	78	25.8	n/s	n/s
BH09/12	64	F	163	113	42.5	n/s	n/s

victim did not drink any alcohol on the day of the incident. Possibly 15 to 30 minutes later, the victim and the same friend re-entered the water. His friend wore his own fins, while the victim borrowed the friend's mask and snorkel as he was keen to try these. He was also wearing shorts. The water was murky and there was a fast current that increased further away from the shore.

After about 15 minutes, the victim began to struggle to stay afloat. His friend grabbed him from behind and tried to support him but was forced to let go because of cramp. The victim submerged temporarily before he re-surfaced, waved his arms, and called "Help". At this point, the river was 70–100 m wide, the current was very strong and the swimmers were being swept downstream. Some bystanders responded to his call and entered the water to assist, some with the aid of an air mattress. However, by the time they reached the victim he had submerged and they were unable to find him. The friend was brought to shore with the aid of the air mattress.

The victim's body was found four days later, well down river. His mask was still present, although displaced, and his body was in a relatively advanced state of decomposition.

Autopsy: The body was decomposed. The heart weighed

160 g and showed decompositional change but was otherwise normal. Severe decomposition and the absence of a record of height and weight means that a valid normal weight for the heart cannot be given. The lungs were not weighed and showed decompositional changes. The upper airways contained gastric contents. The cause of death was given as drowning. Toxicology: blood alcohol 0.123 g 100 ml⁻¹, some or all of which may be owing to decomposition.

Comments: It is easy even for experienced swimmers to underestimate the strength of a current in a river or similar waterway, and deaths commonly occur in such places. For example, in Australia in 2011–12, there were 284 reported drowning deaths. Seventy-five (26%) occurred in rivers, creeks and streams (often involving currents), compared to 55 (19%) at beaches, and 43 (15%) in oceans and harbours.³ It is important to obtain local knowledge about potential hazards although, in this case, the victim and his friends spoke little English, which could have made this more difficult. It is difficult to tell whether the alcohol had been ingested or was generated by post-mortem decomposition.

Summary: Unknown medical history; appeared to be fit and healthy; weak swimmer; inexperienced snorkeller; large river with strong current; struggled to stay afloat; friend

Table 1 (cont.)

diving-related fatalities 2009

GSB – group separated before; n/a – not applicable; n/s – not stated

Dive group	Dive purpose	Depth (msw)	Incident (msw)	Weight belt	Wts (kg)	Disabling injury
BSB	spearfishing	n/s	surface	n/s	n/s	Cardiac incident
GSB	recreation	n/s	surface	n/s	n/s	Cardiac incident
BNS	recreation	n/s	surface	n/s	n/s	Asphyxia
BNS	recreation	n/s	surface	n/s	n/s	Asphyxia? Cardiac incident?
BNS	recreation	n/s	surface	n/s	n/s	Cardiac incident
n/s	recreation	1	surface	n/s	n/s	Cardiac incident?
solo	training	1.5	1.5	n/s	1	Asphyxia? Cardiac incident?
solo	recreation	2	n/s	n/s	n/s	Asphyxia
BSB	spearfishing	12	n/s	yes	n/s	Asphyxia
solo	spearfishing	15	2.4	yes	3	Asphyxia
GSB	recreation	20	surface	n/s	n/s	Asphyxia
GSB	recreation	n/s	surface	no	n/a	Asphyxia? Cardiac incident?

present but cramped and unable to assist; attempted rescue by bystanders; drowning

BH 09/04

This victim, a 71-year-old male, was described by his general practitioner as “*fit and healthy*”. He had suffered from back stiffness resulting from ankylosing spondylitis for the past 30 years but was no longer taking medication for this and was on no other medications. He and his wife swam daily during the summer months at their local ocean beach. He was described as “*not a bad swimmer*” who wore goggles (it is unclear whether these were swim goggles or a mask; both were mentioned in various reports) and a snorkel, as his back condition made it difficult for him to lift his head while swimming.

On this occasion, the victim and his wife were having their regular swim in calm, shallow water. There was no mention of any current. After a short time, as the wife was leaving the water, she heard a strange sound and turned to see her husband removing his goggles and gasping for breath. She went back into the water and, with the assistance of another swimmer, helped the victim onto the beach. A nearby tourist, who happened to be a doctor, began BLS. When paramedics arrived, they found the victim to be

asystolic and they continued resuscitation en route to a nearby hospital. Although he initially appeared to respond to ALS, the victim’s condition deteriorated and he was soon pronounced dead.

Autopsy: The trachea contained a small amount of frothy fluid. The right and left lungs weighed 682 g and 596 g respectively (NR 663 +/- 217 g, 569 +/- 221 g). There was diffuse pulmonary oedema and congestion. The heart weighed 426 g (NR 400 +/- 69 g). There was mild atherosclerosis of the coronary arteries without significant stenosis, mild sclerosis of the mitral valve and minor thickening of the blood vessels in the AV node. The cause of death was given as drowning.

Toxicology: alcohol undetected (limit not stated).

Comments: It is not clear what caused the drowning. Ankylosing spondylitis can have cardiovascular manifestations, which include atrio-ventricular block and bundle branch block and valvular disease.⁴ It is also possible that the victim developed immersion pulmonary oedema, most likely secondary to a dysrhythmia.

Summary: Ankylosing spondylitis; otherwise appeared to be fit and healthy; regular swimmer; swam with goggles and snorkel because of back stiffness; calm and shallow;

became breathless and collapsed; prompt BLS and ALS unsuccessful; drowning

BH 09/05

This 61-year-old woman was an overseas tourist visiting the GBR with her husband. Her husband described her as “*in good health and not taking any medication for a heart complaint*”. However, she was obese (BMI 32.3 kg m⁻²) and was taking an unknown medication for hypertension. Her husband reported that she was a competent snorkeller although she had apparently got into some difficulty while snorkelling in Italy on another occasion. She did not declare any medical conditions to the snorkel operator.

She was with a group of 20 tourists of the same nationality (including an interpreter) on a day tour on a vessel with 88 passengers. The passengers were split into two groups, the victim being part of the first group offered the opportunity to snorkel from an island beach fringed by a coral reef that began about 20 metres from the shore. There was a buoyed line at the extremity of the snorkelling area, beyond which the strength of the current was considered unsafe. A tender was tied to the inside of this line and a lookout was aboard this tender to help snorkellers if required. There was another lookout on the beach. The weather was described as sunny but the surface conditions were unreported. The operator’s staff reported that there was little current initially, but it appears that this quickly changed to become less than 1 knot near the beach increasing to 5 knots further out.

The victim and her husband entered the water from the beach, wearing a mask, snorkel, fins and a full-length stinger suit. After a short time, she returned to shore to get better-fitting fins while her husband swam towards the buoyed rope where fish were being fed from the tender. After changing fins, the victim re-entered the water and, accompanied by another passenger (a weak swimmer who was using a ‘noodle’ buoyancy aid), snorkelled towards the tender. However, when she encountered a strong current she became anxious and began to panic. Her buddy shared his ‘noodle’ with her and soon her husband arrived on board another tender and together they were towed to chest-deep water. The tender then departed to assist other snorkellers caught in the current. In fact, 16 out of the 20 snorkellers needed assistance from the tender owing to the current.

The victim became unconscious soon after walking to shore with the help of her husband and another passenger. The husband assessed her as apneic and pulseless and commenced BLS. He was quickly relieved by three doctors (two of whom were part of the tour group and one who was a guest on the island). A bag-valve-mask was used to provide oxygen-supplemented ventilation; an automated external defibrillator (AED) brought from a nearby boat reported “*No shock advised*” (i.e., the victim was not in ventricular fibrillation or ventricular tachycardia). BLS was continued

for 50 minutes, without success. A rescue helicopter with paramedics arrived but the topography of the island made it too difficult to move the victim to the helipad for evacuation. The doctors pronounced the victim to be deceased.

Despite one of the investigating policemen suggesting that the operator may have been negligent in allowing the snorkelling while such a strong current was present, the Workplace Health and Safety investigation did not support a prosecution, nor did the investigating coroner recommend one. Although the area was known to be prone to strong currents, these were reported to be somewhat unpredictable.

Autopsy: The heart weighed 402 g (NR 362 +/- 77 g). There was severe atherosclerotic narrowing of the ostium of the right coronary artery and mild atherosclerosis of the other coronary arteries. Histology revealed subendocardial fibrosis but no acute ischaemic changes. The right and left lungs weighed 537 g and 469 g respectively (NR 561 +/- 256 g, 491 +/- 204 g). The lungs showed mild to moderate congestion and oedema. The trachea and bronchi contained a small to moderate amount of mucoid exudate. The liver showed fatty change. The cause of death was given as myocardial ischaemia due to right coronary artery stenosis due to atherosclerosis.

Toxicology: (performed on cranial blood, usually femoral venous blood is used) alcohol undetected (< 10 mg 100 mL⁻¹); diethyltoluamide (DEET) present – the significance of this finding is unclear.

Comments: This victim’s cardiac event was likely precipitated by exertion against a strong current and associated anxiety, and could have occurred in a variety of other physical and/or anxiety-provoking situations. It is obvious that the tour operator erred in allowing its passengers to snorkel at that time given the strength of the current that developed and the number of snorkellers requiring assistance. It appears that the current is unpredictable at that site. An operator is well advised to discourage inexperienced and/or obviously unfit clients from snorkelling in such areas. The operator’s staff appears to have been well prepared and responded quickly and appropriately to the circumstances.

Summary: Obesity; hypertension; previous snorkelling experience (including an unknown adverse experience); strong current; collapsed on reaching shore; cardiac death

BH 09/06

This 76-year-old male was an overseas tourist on a tour of the GBR with a group from his country. He had a history of hypertension for which he was prescribed medication (unknown). His wife later reported that he had previously suffered from diabetes mellitus but apparently no longer required medication for this. He did not declare any medical conditions to his travel insurer or to the tour operator on its pre-snorkel medical questionnaire. It appears that the victim

was only a weak swimmer with no previous snorkelling experience. The group had been given a snorkel briefing, translated into the victim's language by an interpreter. In addition, each member was also given a written translation of the snorkel briefing on the day prior to the incident. After this briefing, some of the group went snorkelling. The victim was seen walking in the water and looking down into it through a mask, although he was not seen to be snorkelling as such.

On the following day he elected to go snorkelling with up to five others from his group, under the watch of a lookout. The group entered the water from the sandy beach of an island bay. The weather was described as clear and the sea calm. He was wearing a mask and snorkel, shorts and a t-shirt. Floatation aids had been offered but he had not taken any. He also had not buddied-up with another snorkeller.

The lookout reported that he saw the victim wade into waist-deep water to about 30 metres from shore from where he began snorkelling. About a minute later, the lookout became concerned when he noticed that the victim had not moved and, when the victim did not respond to his calls, the lookout ran into the water and went to him. After shaking the victim without eliciting a response, the lookout rolled him over and found him to be unconscious, pale and apparently apneic, but with his mask and snorkel in place. He quickly towed the victim to shore and placed him into the recovery position, causing a large amount of water and froth to be exuded from the victim's mouth. After checking for, and not finding, a palpable pulse, the lookout and another staff member immediately began BLS. It was estimated that BLS was commenced within four minutes of noticing the victim motionless in the water. Supplemental oxygen was provided and an AED was attached about a minute later but advised that no shock be given. A nurse from the nearby resort arrived and assisted with resuscitation efforts, which were ceased after 25 minutes in accordance with medical advice over the phone.

Autopsy: The heart weighed 427 g (NR 370 +/- 75 g) and showed concentric left ventricular hypertrophy (17 mm). There was greater than 75% narrowing of the LAD and circumflex coronary arteries with scarring of the left ventricle consistent with previous infarction, but no acute ischaemic changes. The right and left lungs weighed 559 g and 500 g respectively (NR 651 +/- 241 g, 579 +/- 201 g). The lungs showed anthracosis, congestion and moderate pulmonary oedema. The trachea and bronchi contained fluid debris. The cause of death was recorded as drowning. Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹); flunitrazepam 0.01 mg kg⁻¹ (this is unusual as this is a fast-acting benzodiazepine used as a sedative).

Comments: It appears likely that this victim drowned as a result of a cardiac arrhythmia due to severe pre-existing ischaemic heart disease. It is interesting to note that, although he was elderly, a poor swimmer and had no snorkelling

experience, he was not paired with a buddy or using a buoyancy aid. Of note also, the police investigator reported that the interpreter spoke very poor English, making it likely that the pre-snorkelling briefing was poorly translated and important advice may not have been relayed. It is unknown if the presence of a buddy and/or the use of a buoyancy aid would have prevented this death. However, both are desirable, especially with an elderly, weak swimmer and inexperienced snorkeller.

Summary: History of diabetes mellitus; hypertension; weak swimmer; no previous snorkelling experience; good conditions; waist-deep water; no buddy; no buoyancy aid; silent unconsciousness; drowning (likely cardiac-related)

BH 09/07

This 31-year-old male was apparently very healthy with no known significant medical history. He was an experienced spear fisherman and underwater photographer and held an Advanced Open Water Scuba qualification. He had been learning some apnea diving techniques to extend his breath-hold capability and had practised these on multiple occasions doing underwater laps in a local swimming pool, each session lasting for approximately an hour. On this occasion, he again came to the pool to practice. The pool, which was 20 metres long with depths varying from 1.0 to 1.6 metres, was located in a gymnasium complex. Although no lifeguard was present, there was a surveillance video camera (non-recording), which was monitored from the reception desk. The victim was wearing mask and long free-diving fins and was carrying a 1 kg weight (described as "looking like a dumbbell") in his hands. He was alone in the pool.

About 3–5 minutes after he was last seen swimming underwater, a witness noticed him motionless, face-down on the bottom against one end of the pool (and out of view of the camera) with a plume of bloody fluid coming from his mouth and nose. After asking someone to call for an ambulance, the witness pulled the victim onto the pool deck and checked his vital signs. He was unconscious and apneic with no palpable pulse. The witness then rolled him onto his side to drain his airway before beginning BLS. This was an estimated five minutes from the time the victim was last seen swimming. When paramedics arrived approximately five minutes later, they found the victim in asystole and implemented ALS, which continued for approximately 30 minutes. Spontaneous circulation returned for a short period although the victim remained apneic. He was transported to hospital but died shortly afterwards.

Autopsy: The heart weighed 417 g (NR 370 +/- 75 g) and appeared normal. There was no coronary artery disease. Sections from the artery to the AV node showed fibromuscular dysplasia. The lungs were heavy and showed widespread oedema, suggestive of drowning. The right and left lungs weighed 1,014 g and 868 g respectively

(NR 651 +/- 241 g, 579 +/- 201 g). There was a small amount of fluid in the trachea and bronchi. Histology of the lung showed focal pneumonitis. The cause of death was given as drowning.

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: This appears to be another case of a healthy, experienced breath-hold diver failing to appreciate the potential dangers of extended apnea, even when practised in the confines of a shallow pool. Doing so alone without the guarantee of immediate rescue proved fatal. However, the significance of fibro-muscular dysplasia of the artery to the AV node is controversial, so a cardiac arrhythmia related to this cannot be excluded. Autopsy alone does not exclude conditions such as long QT, Brugada syndrome or catecholaminergic polymorphic ventricular tachycardia.

Summary: Apparently very fit and healthy; fibro-muscular dysplasia of artery to AV node seen at autopsy; experienced breath-hold diver; alone in pool pushing breath-hold limits while swimming repetitive underwater laps; drowning (likely due to apneic hypoxia although cardiac arrhythmia possible)

BH 09/08

This overseas tourist, a 28-year-old male, was a social drinker and moderate smoker who, apart from suffering haemorrhoids and myopia, was reported by his family to be a healthy young man. He was a non-swimmer and had used a mask and snorkel for the first time two days earlier while standing in shallow water watching fish.

On this day, he and two friends went to a popular snorkelling area off a sandy beach. The weather was described as warm and sunny and the visibility was up to eight metres. Although the area is prone to strong, long-shore currents, the current was described as weak at the time. The friends snorkelled for a while and the victim, wearing his newly-purchased mask and snorkel, stood in waist-deep water with his face on the surface and watched fish through his mask. He was wearing only board shorts and had no floatation aid. After about 45 minutes the three returned to the beach. The others wanted to go for a walk and the victim decided to re-enter the water alone. Approximately 10 minutes later, the friends heard calls for assistance and when they walked back along the beach saw rescuers performing BLS on their companion.

Another snorkeller had noticed the victim motionless on the seabed at a depth of 1.8 msw about 15–20 metres from shore. His mask was about two metres away but his snorkel was not visible. The snorkeller prodded the victim with her foot and, after there was no response, called for help and was soon joined by her brother. Together they dragged the victim onto the shore. When they placed him in the recovery position, water and froth exuded from his mouth. He was apneic and apparently pulseless (the radial pulse was checked although this is a poor indicator of absence of circulation in such a circumstance) so BLS was commenced

by the rescuers and bystanders. Ventilations were continued despite the outflow of copious amounts of froth and water. One witness described what appeared to be a “purple jellyfish” coming from the victim’s mouth. Resuscitation was continued for 60 to 90 minutes. At one point, rangers arrived with oxygen equipment. When paramedics arrived more than an hour later, the victim was found to be asystolic and was pronounced dead.

Autopsy: The brain was large and heavy 2,002 g (NR 1,449 +/- 161 g) with prominent cerebellar tonsils consistent with an Arnold-Chiari type 1 malformation and megaloccephaly. The heart weighed 392 g (NR 370 +/- 75 g) and was normal with no coronary atherosclerosis. The lungs were very heavy and oedematous. The right and left lungs weighed 1,508 g and 1,392 g respectively (NR 651 +/- 241 g, 570 +/- 201 g). The upper airways contained fluid. The cause of death was given as drowning.

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: Individuals with abnormalities such as the Arnold-Chiari malformation (in which there is downward displacement of portions of the medulla and cerebellum into the cervical spinal canal) have reported increased incidents of sleep apnea and are at risk of sudden death from brainstem compression. Indeed, sudden death may be the first indication of an underlying Arnold-Chiari malformation. There have been a number of reported cases in childhood (ages from 17 months to 17 years) of sudden death in the absence of pre-existing neurological deficits.⁵ It is not possible to tell if the brain pathology caused the drowning, but the forensic pathologist who performed the autopsy believed that it was likely. Alternatively, this victim was a non-swimmer who was alone at the time of the accident. The seabed in the area can become steep and there was a current and it is possible that he stepped into deeper water and panicked, possibly removing his mask and snorkel, and silently drowned.

Summary: Asymptomatic Arnold-Chiari malformation; non-swimmer; non-snorkeller; no buoyancy aids; standing alone in waist-deep water wearing mask and snorkel looking at fish; alone; possible sudden death from brain malformation or stepped into deeper water; drowning

BH 09/09

This victim was a healthy 31-year-old non-smoker with no significant medical history. He was a good swimmer and was certified as an Assistant Scuba Instructor. He was also a highly-experienced spear fisherman who did so regularly, reportedly diving every weekend. He had been diving for seven years and was president of a local dive club.

He set off spearfishing with three friends with whom he had dived with often, although it was the first time he had been to this site. The weather was fine and sunny, a light wind and calm sea, a one-metre swell and some surge. The water

temperature was around 15°C and visibility varied from 3–10 metres. The victim was wearing a mask and snorkel, long fins, a lycra suit underneath a full-length wetsuit with hood, an inner vest with some weights, and outer wetsuit vest, bootees and gloves. He had a weight belt with three weights and carried a speargun with 30 m of line and a float with flag. All of the others also had floats with flags. They dived as two buddy pairs, with each diver generally about 50 m from another, although sometimes as far as 100 metres.

After a little over 2 hours the other pair of divers left the water. The victim's buddy exited 15 minutes later, noticing his friend's buoy bobbing in the water at that time and presuming that he was fine. He had last spoken to the victim about 30 minutes earlier, comparing their catches. However, a while later, the group became concerned when they noticed that the float (which was 700–800 m from shore) had not moved for some time and they could not see any sign of their friend. They alerted a jet skier who went to the float. He reported back that he could see the speargun on the seabed but saw no sign of the victim. The police were called and a search was begun, initially by people at hand and shortly afterwards by the police. The victim's spear was soon retrieved, embedded in what was described as a 2.5 m wobbegong shark. The victim's body was found the next day lying face-up on the seabed at a depth of 12 metres' sea water (msw), adjacent to a wall rising to a rocky ledge 3–4 m from the bottom. His mask, fins and weight belt were in place but his snorkel was out of his mouth.

Autopsy: The autopsy was performed two days after death. The heart weighed 320 g (NR 400 +/- 69 g) and was normal with up to 10% narrowing of the coronary arteries by atherosclerosis. There was blood-tinged frothy fluid in the lower airways. The lungs were moderately expanded and heavy. The right and left lungs weighed 760 g and 660 g respectively (NR 663 +/- 217 g, 569 +/- 221 g). The cause of death was reported to be drowning (possibly subsequent to "shallow water blackout").

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: It is impossible to know what caused this victim's demise as it was unwitnessed – yet another failure of the 'buddy system'. It is possible that he became unconscious as a result of apneic hypoxia. There were no reports indicating whether or not he practised hyperventilation. It is also possible that he got dragged underwater by the shark and subsequently drowned.

Summary: Healthy; assistant scuba instructor; regular and experienced spear fisherman; poor buddy system; 2.5 m shark on spear; drowning (apneic hypoxia?)

BH 09/10

This 20-year-old male, a highly accomplished surf lifesaver and ironman who was extremely fit and healthy, had been spearfishing infrequently for about two years. He joined

a group from a local spearfishing club on a five-day spearfishing trip on a large live-aboard vessel on the GBR. At an initial safety briefing, they all signed a disclaimer which, among other things, warned of the dangers of 'shallow water blackout' and the potential for hyperventilation to increase the likelihood of it occurring.

The 13 passengers dived in groups of three or four, each having access to a tender with outboard motor and hand-held radio. They also had access to a crew member to drive it if they so wished. The victim buddied up with two others for the trip. He wore mask, snorkel and fins; a full-length, two-piece 4 mm wetsuit with hood; a weight belt with two 1.5 kg weights, gloves, bootees and a dive knife. He carried a float with 25 m of line which was attached to his spear gun.

The second day, one of his companions noticed that he would lie on the seabed at 22 msw for up to about two minutes waiting for fish to swim by. The victim later mentioned that he was able to dive to 22 msw for two minutes and 10 seconds. The companion expressed concern that this technique was risky owing to "not being aware of your depleting oxygen stock" and the victim agreed that he would avoid doing this and would not dive beyond 25 msw without support from another diver. The companion also noted that the victim spent very little time on the surface between dives. The other companion also noticed the short surface intervals but was impressed with the victim's breath-holding capabilities and ability to reload his speargun underwater without surfacing.

On the third day, the victim and his companions set off again in their tender, without a crew member on board. He was apparently well, but mentioned that he had sore ears from equalisation problems the day before. The weather was described as clear with a light wind, waves of less than one metre, good visibility and a water temperature of 22°C. The trio drifted in the current in water of depths from around 10 to 14 msw with the unmanned tender drifting behind. At times they were up to 100 m apart. After approximately three hours, the other divers boarded the dinghy and saw the victim's float 50 to 70 metres away. They watched the float for about a minute, becoming concerned when it appeared to be dragging and their companion had not yet surfaced.

One of the companions re-entered the water and, when she swam down the line, saw the victim lying on his back on the bottom, apparently unconscious. He was lying about 10 m from his spear, which was embedded in the reef. She surfaced and called for help before diving down to the victim, who was at a depth of 14.6 msw, releasing his weight belt and bringing him to the surface. A plume of blood came from his mouth. The two companions dragged the victim into the tender and removed his mask which was half-filled with blood. There was "a lot of bright arterial blood and bloody foam coming from his mouth and nose". One companion gave several rescue breaths, despite the continued outflow of blood-stained, frothy sputum with each breath. They

radioed the main vessel to report the problem. After checking for and finding no palpable pulse, one of the companions began BLS while the other drove the tender to the main vessel. The victim was brought aboard the main boat and BLS was continued and supplemental oxygen provided with ventilations via a bag-valve-mask. The skipper had alerted the Royal Flying Doctor Service (RFDS) and BLS was continued for at least an hour with telephone consultation with the RFDS doctor, who then advised that efforts be abandoned given a submersion of at least eight minutes and the lack of response to BLS.

The victim's dive watch/computer indicated that he had performed 68 breath-hold dives that day. The final dive was to a maximum depth of 14.9 msw for a total time of more than eight minutes. The record indicates that he began to ascend to the surface after one minute and 44 seconds but, after reaching a depth of 2.1 msw, appears to have sunk to the bottom (14.6 msw) over a minute and remained there for almost six minutes before being rescued.

Autopsy: There were numerous petechiae on the conjunctiva of both eyes (possibly the result of mask squeeze). The heart weighed 545 g (NR 400 +/- 69 g) with symmetrical left ventricular hypertrophy. There was no histological evidence of hypertrophic cardiomyopathy, and the coronary arteries showed no atherosclerosis. The trachea and main bronchi contained bubbly haemorrhagic fluid. The lungs were very heavy and waterlogged. The right and left lungs weighed 1,400 g and 1,420 g respectively (NR 663 +/- 217 g, 569 +/- 221 g). The cause of death was given as drowning (possibly subsequent to "shallow water blackout").

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: The heart was heavy but this is consistent with the so-called 'athlete's heart', i.e., work hypertrophy associated with high-level athletic training such as 'ironman' training. This condition can be associated with high vagal tone and bradycardia. The finding of petechiae on the conjunctivae is unusual in this reviewing pathologist's (CL) experience. It could be the result of mask squeeze but is described in up to 5% of natural deaths.⁶

It is likely that this victim died while trying to free his embedded spear from the reef. With a documented breath-hold dive of almost two minutes, coupled with exertion from trying to retrieve the spear, it is likely that he succumbed to apneic hypoxia shortly before reaching the surface. As a result of a 'loose' buddy system, he was unconscious and apneic underwater for around six minutes in relatively warm waters, resulting in a poor prognosis for survival. It is unknown whether or not he practised hyperventilation prior to breath-holding.

Summary: Very fit and healthy; accomplished surf lifesaver and ironman; experienced spear fisherman; poor buddy system; spear embedded in reef; drowning (apneic hypoxia)

BH 09/11

This 68-year-old male was reported to have been healthy other than suffering from reflux, for which he took pantoprazole. He was on a day trip on a large sightseeing vessel which visited several islands of the GBR. He did not declare any medical conditions on the pre-snorkel questionnaire. He was reported to be a competent swimmer but there is no indication whether he had previous snorkelling experience. His wife described him as looking pale and unwell that morning.

After a briefing, the group was provided with snorkelling equipment, including an offer of vests or 'noodles' as buoyancy aids. The victim did not take a buoyancy aid. Some of the group then entered the water to snorkel. There were 37 passengers but there is no indication of how many of these snorkelled. The victim was wearing board shorts, a t-shirt and a mask and snorkel but there is no mention whether or not he was wearing fins. There were two lookouts on the main boat. The conditions were described as calm with a light wind. There was a current, which one crew member later reported as 2 knots, but it must have been beyond where most of the group were snorkelling as most had no problem with the current. They were briefed on the designated snorkelling area, which was up to 20 msw deep.

The victim was seen by one of the lookouts shortly after entering the water. He was dog-paddling towards the boat but then put his face down and snorkelled with his arms beside him. He was not noticed again. However, when the group returned to the vessel some 45 minutes later, a head-count revealed that one person was missing and a search was commenced. The victim was found about 10 minutes later floating face down on the surface 50–80 m from the vessel. He was unconscious and apneic. A person on the tender quickly gave him several rescue breaths, dragged him aboard and began BLS during transfer to the main vessel. BLS was continued with ventilations now being provided using a bag-valve-mask and supplemental oxygen. The victim regurgitated water and other stomach contents. When the boat arrived at an island about 45 minutes later, it was met by the island nurse who reported the victim to be cyanotic with mottled skin and fixed, dilated pupils. She inserted an oropharyngeal airway and attached a defibrillator but no shock was given. After several more minutes of BLS, efforts were abandoned and the nurse declared the victim to be dead.

Autopsy: The brain was heavy (1,629 g; NR 1449 +/- 161 g). The trachea and main bronchi contained a large amount of frothy, pink, watery fluid. The right and left lungs weighed 583 g and 571 g respectively (NR 663 +/- 217 g, 569 +/- 221 g) and showed moderate oedema and large amounts of frothy fluid on the cut surface. The heart weighed 334 g (NR 400 +/- 69 g) and was normal with mild atherosclerosis of the coronary arteries. The cause of death was given as drowning.

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: The victim's prior snorkelling experience is unknown but the lookout's report of him "dog-paddling" towards the boat does not inspire confidence that he was competent. It is unknown whether he got into difficulties within the designated snorkelling area or if he swam or was carried outside it by the current. In any case, it is evident that the lookouts were ineffective, this being unsurprising if a large number of the passengers were in the water at once. He might have been feeling unwell and this could have been exacerbated by the snorkelling, or he might have simply drowned as a result of inexperience.

Summary: Apparently healthy; looked unwell in morning; reflux on pantoprazole; competent swimmer; unknown snorkelling experience; likely many snorkellers in water; lookout failure; drowning (silent?)

BH 09/12

This 64-year-old woman was an overseas tourist who was visiting the GBR with her husband. She was morbidly obese (BMI 42.5 kg m⁻²) and had an extensive medical history including asthma and allergies, hypertension, depression, hysterectomy, cataract removal, bladder repair and InterStim[®] therapy. She was taking a variety of regular medications including diltiazem, venlafaxine, pregabalin, clonazepam, ezetimibe, colesvalam, oestradiol, esomeprazole and an undisclosed, injectable asthma/allergy medication.

The victim was a poor swimmer who had only very recently taken lessons. Her snorkelling experience is unknown. The operator gave a brief to the approximately 20 prospective snorkellers. This included information on the use of snorkelling equipment, hand signals, the buddy system and also the requirement to report any health conditions to staff. The victim did not declare any conditions or medications on the written medical declaration and waiver form or report her health problems.

At the site, the conditions were described as clear with a moderate wind (10–15 knots), calm and with little current. Before he went off scuba diving, the victim's husband checked his wife who said she was fine. The victim was wearing mask and snorkel and a stinger suit and carried a 'noodle' floatation device. She entered the water and was soon seen by the lookout snorkelling alone about 6–8 metres from the boat. However, about five minutes later crew members became concerned to see her motionless and apparently unconscious, about four metres from the stern. No-one had heard any splashing or call for help. One crew member entered the water and towed the victim to the boat. Once on board, she was found to be unconscious, apneic, and with no palpable pulse. BLS was commenced by the crew and supplemental oxygen was provided. An AED was attached but it appears that no shock was given. BLS was continued for about 50 minutes until the arrival of paramedics, who soon after declared the victim to be dead.

Autopsy: The trachea and major bronchi were clear of fluid. The right and left lungs weighed 519 g and 584 g respectively (NR 561 +/- 256 g, 491 +/- 204 g) and were not oedematous. The heart weighed 434 g (NR 362 +/- 77 g) There was mild left ventricular hypertrophy (16 mm; NR < 14 mm). There was less than 10% narrowing of the coronary arteries by atherosclerosis and some focal myocardial scarring, contraction bands and some luminal narrowing of the intramyocardial arteries. Serum tryptase was mildly elevated at 43 µg L⁻¹, but mild elevations of post-mortem tryptase are common and of questionable significance. Death was attributed to drowning (with obesity-associated heart disease and asthma). *Toxicology:* ephedrine/pseudoephedrine chlorpheniramine, clonazepam and diltiazem in their therapeutic ranges; venlafaxine 1.5 mg L⁻¹ (which is in the potentially toxic range, although this drug is subject to post-mortem redistribution); alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: Had this victim declared her various medical conditions to the operator, it is likely she would have been strongly advised against, or possibly prevented from snorkelling, especially without being accompanied. However, prospective snorkellers and divers are often reluctant to declare potentially adverse health conditions for fear of being prevented from undertaking their chosen activity. After an investigation by Workplace, Health and Safety (WHS), the operator was advised to strengthen its warnings during the briefing about the impact of certain medical conditions on snorkelling safety.

This silent drowning could have been precipitated by a cardiac dysrhythmia, asthma or simply by aspiration causing laryngospasm and sudden unconsciousness. Cardiac fibrosis has been associated with heart failure and arrhythmias. The combination of drugs, particularly anti-arrhythmics, anti-convulsants and anti-depressants, especially in conjunction with pseudoephedrine and underlying myocardial changes, may well have been lethal. Finally, the victim was a poor swimmer. If this had been known to the operator, she should have been closely supervised. Routine questioning about swimming and snorkelling experience and ability is always advisable, accepting that answers must be taken on faith.

Summary: Morbid obesity; asthma, hypertension, depression, InterStim[®] therapy, multiple medications; poor swimmer with unknown snorkelling experience; reported to be well that morning; lookout but no buddy; silent drowning (?cardiac-related)

Scuba diving fatalities (Table 2)

SC 09/01

This fit-looking, 47-year-old male dived for crayfish or abalone regularly, usually alone. He was certified as an Advanced Open Water Diver 20 years earlier. When undergoing a pre-employment medical, he reported to the

Table 2

Summary of scuba and surface-supply diving-related

BNS – buddy not separated; BSB – buddy separated before; GSB – group separated before; SC – scuba; SS – surface-supply breathing apparatus

ID	Age (yr)	Gender	Height (m)	Weight (kg)	BMI (kg m ⁻²)	Training	Experience	Dive group
SC09/01	47	M	180	91	28.1	trained	experienced	solo
SC09/02	17	M	180	71	21.9	trained	some	BSB
SC09/03	20	F	n/s	n/s	n/s	in training	nil	GSB
SC09/04	59	M	183	81	24.2	trained	some	GSB
SC09/05	50	M	170	76	26.3	trained	experienced	GNS
SC09/06	52	M	186	107	30.9	trained	experienced	GSB
SC09/07	53	M	178	73	23.0	trained	experienced	BNS
SC09/08	33	M	170	57	19.7	trained	some	GNS
SS09/01	36	M	181	85	25.9	trained	some (not commercial)	solo

doctor that he had a history of epilepsy but had been fit-free for many years. The doctor subsequently gave him several repeat prescriptions for phenytoin 100 mg, 4 nocte. The doctor who performed the pre-employment medical, and subsequently prescribed phenytoin, had no training in diving medicine. He was unaware that the victim was a diver so had not discussed this with him.

On this day, the victim set out to dive alone at a well-known shore dive site, predominated by rocky reef and kelp and which was subject to a large swell and surge. Prior to going, he called a friend to enquire about the conditions and was told “*it’s not ideal but you’ve certainly dived in worse conditions so it should be OK*”. He was wearing a full-length wetsuit with hood, mask, snorkel, fins, knife, BCD, 12.2 l tank (WP 232 bar) and a pocket-style weight belt with seven weights (17 kg), booties and gloves. He had a catch-bag attached to his BCD by a clip.

Searchers saw his body the next morning about 10 metres from shore. He was floating in shallow water with waves washing over him. His yellow fins and his arms were visible on the surface. They waded out to him and found that one of his hoses (HP hose) was wedged between rocks and so they removed his BCD and tank and towed him to shore, leaving the equipment in the water. When later recovering this, they cut the snagged hose in order to free it. Air immediately began to escape but the searcher quickly turned off the valve.

When later inspected, the demand valve’s mouthpiece was reported to have been in good condition (despite what was described as a “*5 mm cut on its underside*”). When tested, the regulator was found to be “*acceptably functional*”. The inflate/deflate hose was missing from the BCD which consequently could not be inflated. Although out of test, the cylinder was in good condition and, when checked, held a pressure of 160 bar and the air met relevant purity standards.

Autopsy: Radiology revealed no arterial gas embolism. There was focal bruising on the left chest and right thigh. The heart weighed 425 g (NR 400 +/- 69 g) and the left ventricle measures 15 mm in thickness. There was no gas in the heart. There was mild atherosclerosis in the coronary arteries. The lungs were well inflated with moderate to severe oedema. The right and left lungs weighed 1,010 g and 876 g respectively (NR 663 +/- 217 g, 569 +/- 221 g). There was froth in the trachea and bronchi. The brain weighed 1,525 g (NR 1,449 +/- 161 g) and appeared normal. Neuropathological examination revealed only cerebral congestion. Analysis of the vitreous revealed sodium of 150 mmol L⁻¹ and an osmolality of 350 mosmol L⁻¹ and such levels have been reported to be associated with drowning.⁷ The cause of death was reported to be drowning. (The forensic pathologist suggested that epilepsy could have contributed to the drowning.) Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹); cannabis 5 µg L⁻¹; 2% carboxyhaemoglobin (probably from smoking); phenytoin 7 mg L⁻¹.

Table 2 (cont.)

fatalities in Australian waters in 2009

GNS – group never separated; GSD – group separated during; + sufficient air (to surface safely); ++ 1/4–1/2 full tank; +++ >50% full; nad – no apparent defects; n/i – not inflated; n/s – not stated

Dive purpose	Depth (msw)	Incident (msw)	Weight belt	Wts (kg)	BCD	Remaining air	Equipment test	Disabling injury
n/s	n/s	n/s	on	17	n/i	+++	some issues	Asphyxia
recreation	8	surface	on	7–8	n/s	++	nad	Asphyxia? CAGE?
training	3	surface	n/s	9.5	n/s	n/s	some issues	Unknown
recreation	14	surface	on	n/s	n/s	+	nad	Cardiac incident
training	2	surface	on	n/s	inflated	+++	nad	Cardiac incident
recreation	5	n/s	off	20	n/s	+++	CO ₂ 500 ppm	Cardiac incident
recreation	25	surface	on	6	inflated	+	CO ₂ 559 ppm	Cardiac incident? CAGE?
crayfish	17	surface	on	3	n/s	+	CO ₂ 500 ppm	Asphyxia
commercial	8	surface	off	n/s	n/s	+++	CO ₂ 500 ppm	Cardiac incident? CAGE? Seizure?

Comments: Although this victim had a history of epilepsy, it is not known whether or not this contributed to his demise. Although he had reportedly been seizure-free for many years, seizures can recur.^{8,9} A possible clue lies in the ‘cut’ in his mouthpiece, which could have been caused by his biting hard on this during a seizure. Diving alone, especially with a history of epilepsy, is ill-advised. Alternatively, it appears that the sea conditions might have been challenging and, although he was an experienced diver, it is possible that he was thrown against rocks and drowned as a result of impact and/or loss of the regulator. Given the amount of remaining air, especially in light of the loss of air when the hose was cut, the incident likely occurred early in the dive.

Summary: History of epilepsy on phenytoin; experienced solo diver; diving alone for crayfish/abalone; likely poor sea conditions; body found next day; plenty of air in tank; drowning (secondary to seizure?)

SC 09/02

This victim was a 17-year-old male who was apparently fit and healthy, on no medication except griseofulvin for a fungal foot infection. He was a good swimmer who had qualified as an Open Water Diver two years earlier but had logged little more than four hours’ diving since.

He and a friend went diving for abalone from the beach in a small rock-fringed bay. The victim had snorkelled at this site on the day before and was reportedly “*in good spirits*”.

He was wearing a mask, snorkel, fins and a knife, a full 5 mm wetsuit and gloves as well as hired scuba equipment including BCD, dive computer and weight belt with about 7–8 kg of weights. The conditions were described as “*not ideal for diving*”. There was a one-metre swell and also some surge in shallower water with visibility reported to be 10 metres and the water temperature 24°C.

Initially they snorkelled for about 20 minutes before changing to scuba and descending to about 8 msw where the bottom was rocky with some weed. After about four minutes, the victim noticed that he had lost his knife and signalled to his buddy to surface. After a short conversation on the surface they agreed to dive again, and re-descended. The victim, towing a flagged float and carrying the catch bag, swam closely behind his buddy who reported that he was unable to see the victim a short time later. After waiting a minute, the buddy surfaced to try to locate the float, which he was initially unable to see because of the swell. However, he then located it about 50 m away and swam over to it, expecting to find the victim when he pulled on the attached line. Unfortunately, the victim was not there so his buddy swam over to some rocks 50 m distant. He sent a companion, who was snorkelling nearby, for help before removing his gear to climb to higher ground to look for his friend. Unsuccessful, he donned his equipment and began an underwater search. After about 15 minutes, he located his friend under a rocky ledge at a depth of about 6 msw, lying face-down on the seabed. He was unconscious, his mask was full of bloody water and his regulator was out of his mouth.

The buddy inflated the victim's BCD and brought him to the surface. There he removed the victim's mask (and probably his weight belt). The victim was cyanotic and there was a lot of blood in his mouth. The buddy could not detect a palpable pulse and towed his friend to the rocks. Two bystanders began BLS, repeatedly rolling the victim onto his side to drain the blood and water coming from his mouth and nose. BLS was continued by a police officer and a search and rescue officer for about 30–40 minutes during which time an AED was attached, but no shock was advised. An ambulance paramedic then arrived and pronounced him dead.

The victim's dive computer record indicated that, four minutes into the dive, he ascended to the surface from a depth of 4.5 msw. The 'fast ascent' warning was activated, indicating that he had exceeded the recommended 10 msw minute⁻¹ (it appears that the ascent rate was likely 15–20 msw minute⁻¹). He then descended slowly over about 9 minutes to a maximum depth of 8 msw before ascending directly (possibly at a rate of 15–20 msw minute⁻¹) to the surface. He remained on the surface for about a minute before descending rapidly to a depth of 6 msw where he remained motionless for 75 minutes before he was brought quickly to the surface by his buddy.

When checked, his pressure gauge indicated 135 bar of air in his 12.2 L cylinder. Prior to the dive it had read 190 bar, indicating that his air consumption was not excessive. When later tested (both in the workshop and on a dive), no significant faults that could have contributed to his death were found with the equipment used by the victim. The air met relevant purity standards, although the cylinder later failed 'test' owing to internal corrosion.

Autopsy: A CT scan two days after death showed features indicative of gas embolization. There was subcutaneous emphysema in the anterior mediastinum and 20 ml of gas and frothy blood in the aorta and left ventricle. The heart weighed 344 g (NR 342 +/- 58 g) and was normal apart from the gas. There was no coronary atherosclerosis. There was widespread frothy fluid within the airways. The lungs were oedematous and the right and left lungs weighed 928 g and 940 g respectively (NR 670 +/- 249 g, 593 +/- 224 g). The report stated: "*Upon the free margin of the left upper and lower lobe, between the lung lobes, very superficial disruption of the pleural surface in the order of 6–8 mm in maximum depth ... was present in association with small bullae formation.*" There was frothy fluid tinged with blood in the trachea. The cause of death was given as drowning secondary to pulmonary barotrauma/cerebral arterial gas embolism (PBT/CAGE).
Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: What caused this inexperienced diver to ascend is unknown. His equipment was functional and he had plenty of air. It is evident that he surfaced for a short time before becoming unconscious, probably as a result of PBT/CAGE, and then sinking. Although the investigating police officer

reported that the victim ascended very rapidly to the surface prior to sinking quickly back down, the computer record indicates that this ascent was probably at a rate approaching 20 m minute⁻¹ which, although faster than recommended, does not necessarily indicate a panicked ascent. Presuming that the diver then became unconscious, the subsequent rate of descent indicates that he was likely over-weighted. It is unfortunate that he did not inflate his BCD or ditch his weights to enable him to remain on the surface where he would have been found far sooner. Given the submersion time it is not surprising that resuscitation was futile.

Summary: Healthy; good swimmer; inexperienced diver; buddy separation; became unconscious after ascending to surface and sank to bottom; drowning (subsequent to CAGE?)

SC 09/03

This victim was a 20-year-old female, apparently healthy foreign national who was studying in Australia. She was described as an "*inexperienced swimmer*". She and some friends booked on an introductory scuba dive. At the dive shop, she completed the required paperwork and did not declare any medical conditions. She was fitted with a wetsuit. A dive briefing (in English) was given during the 20-minute boat trip to the dive site. Once at the site, the victim was in a group of four with her friends, under the supervision of one of two instructors.

The 'students' entered the water and floated on the surface supported by their BCDs, holding on to a rope at the boat's stern. The victim was wearing a mask, snorkel, fins, 5 mm wetsuit and reportedly a weight belt with 9.5 kg of weights. The group swam on the surface to shallow water near the shore of a small island where they were taught basic skills while standing or swimming in water of a depth of around 1.5 msw. The sea was reportedly choppy and waves disrupted the training from time to time. The visibility was reported to have ranged from 1 to 1.5 metres, described as "*cloudy from recent rains*", and there was a current. When satisfied with their skills, the instructor led the group into deeper water to a depth of around 2.5 msw. The students "*crawled along the bottom*" in a line, with the instructor just ahead, reportedly checking them regularly. The instructor could see the faces of the students but not their entire bodies. After one check, the instructor noticed that the victim was missing and ascended to the surface with the rest of the group. The instructor then called to the boat driver that a diver was missing, told the students to swim to the shore and then began a search.

Prior to the group surfacing, witnesses saw the victim surface alone, call for help and then apparently sink. The lookout jumped into the water to find her but was unable to see her in the cloudy water. The other instructor located the victim on the seabed about 40 minutes later. She was unconscious and apneic with the regulator still in her mouth. He brought her to the surface and onto a police boat where BLS was

performed by police for 15–20 minutes on the way to the boat ramp. There, ambulance paramedics took over resuscitation and continued en route to hospital, where the victim was pronounced dead on arrival.

On inspection the regulator mouthpiece was found to be perforated, potentially enabling water aspiration.

The instructor was charged with manslaughter and, at the committal hearing, representatives for the police and WHS, among others, argued that the victim was over-weighted, that her regulator was poorly maintained and that the instructor erred in taking a group of four totally inexperienced divers diving in such poor visibility. However, the magistrate determined that the evidence was insufficient to support charges of unlawful killing and the charge was dismissed.

Autopsy: Unavailable.

Comments: This was likely a very avoidable tragedy. Although the standards under which the instructor was operating allow a maximum ratio of four students to one instructor on this programme, the instructor is advised to reduce this ratio in the event of adverse conditions, including poor visibility, rough water and/or current. Had this instructor done so, it would have been easier to monitor fewer students. The conduct of such a dive with the instructor swimming in front of the students can increase the likelihood of separation. Introductory dives are better conducted with participants holding hands, linking arms, or by swimming in formations that enable the instructor to see all of the divers at all times. It appears that the student was likely to have been over-weighted and this would have made it difficult for her to remain on the surface without inflating her BCD and/or ditching her weight belt, something that she would have had little or no training in. Even if she had been told about weight-belt ditching, it might well not have been absorbed, given all else that was happening, possibly magnified by a language problem.

Although the actual timings were unavailable to these reviewers, it would be interesting to know how much time was spent in the shallows learning and practising the basic skills prior to setting off on the actual dive. It seems that it might have been minimal. It is important to provide adequate time to enable the students to learn and practice the essential skills and to feel comfortable enough to participate further. Language difficulties may also have affected the briefing and training adversely in this instance. Finally, if the perforation in the mouthpiece caused the victim to aspirate water, it would likely have increased anxiety.

Summary: Apparently healthy; weak swimmer; introductory scuba dive; some chop and current and poor visibility; ratio of four students to one instructor; relatively little training/orientation time; over-weighted; perforation in mouthpiece; separation; probable drowning or CAGE

SC 09/04

This 59-year-old male was an overseas tourist who went on a day trip on a large dive charter boat to dive on the GBR. There were 100–120 passengers on board. He had been certified as an Open Water Diver three years earlier and had logged a total of 30 dives. He appeared to be healthy and, although his medical history remains unknown, he did not declare any medical conditions on the pre-dive medical questionnaire. The victim was grouped with two other divers and was provided with equipment, including wetsuit, BCD, regulator with ‘octopus’, dive computer, 8 L tank (filled to a pressure of 200 bar), and weights. The sea was reported to be calm and visibility about 15–20 metres. No mention was made about the presence or strength of any current.

The group entered the water and the victim and one buddy descended to a maximum depth of 14 msw and waited for four minutes for the other diver to join them. When he failed to do so, they surfaced briefly before re-descending. They met the other buddy underwater and dived for about 15 minutes before the victim lost sight of the others and surfaced again. His buddies soon surfaced and the three descended again and dived for another five minutes before the victim again surfaced. His buddies ascended to find him holding onto the boat’s mooring line and breathing from his ‘octopus’. He told them that he was “OK”, and did not appear to be in any distress, but he wanted to swim back to the boat (60–70 metres away) on the surface. The divers signalled to the lookout on the boat that everything was okay. After one of them (an off-duty paramedic) checked the victim’s gauge (which read 100 bar) and helped to place the victim’s primary regulator into his mouth, the others descended to return to the boat underwater.

The victim was initially seen to swim ‘freestyle’ for a few metres before rolling onto his back and finning towards the boat. The lookout anticipated that it would take the victim about five minutes to reach the vessel. However, after a while the lookout became concerned that the victim was no longer making headway and was not responding to his calls to confirm he was fine. When the victim was about 25 m from the boat, he did signal that he was okay, but now appeared to be swimming aimlessly and making no effort to return to the boat. In fact, he then turned and began to swim away from it.

On returning from the dive (approximately 15 minutes after leaving the victim), one buddy expressed concern that the victim was not on board, prompting the crew to send a tender to retrieve the now partly submerged victim. When approached by the tender driver, the victim was floating on his side, unconscious, with a grey appearance and foamy discharge coming from his nose. The tender driver dragged him aboard and he was taken to the boat where he was confirmed to be apneic and cyanotic with dilated pupils and frothy sputum coming from his mouth and nose. BLS was commenced by one of the buddies (the paramedic), variously

assisted by an ex-nurse, some crew and a passenger who was a doctor. The victim was rolled onto his side several times in order to clear regurgitated material from his airway. After about 50 minutes, a rescue helicopter arrived and a doctor and paramedic were winched onto the boat. When a defibrillator was attached, it indicated that the victim was asystolic and resuscitation was ceased shortly after.

There was 50 bar of air remaining in his tank and the valve was noted to have been turned on fully. When later tested by the police, the equipment was found to be in good working order and the remaining air met appropriate purity standards.

Autopsy: A CT scan performed 6 hours after death showed “*intravascular gas is noted in the cerebral vessels... There is fluid in both maxillary sinuses and both sphenoidal sinuses. There is fluid noted within the trachea and in the main bronchi. There is extensive air space shadowing throughout both lungs consistent with the presence of fluid in the alveoli. There is gas noted within the pericardium. Intravascular gas is noted in the legs and the vessels in the liver as well as throughout the abdomen.*” The gas pattern could be early decomposition or post-mortem decompression artefact.

The heart weighed 349 g (NR 370 +/- 75 g). There was some calcific thickening of the margins of one of the mitral valve leaflets but no vegetations or areas of perforation were identified. The coronary arteries showed severe focal calcific atheroma, with greater than 75% luminal narrowing of the left main, the mid-course of the LAD and right coronary arteries. Histology showed some oedema and wavy fibres but no acute infarction. There was some focal scarring around the AV node. The trachea and major bronchi showed unremarkable mucosal surfaces but contained frothy fluid. The lungs appeared to be hyper-inflated, with contact between the medial aspect of the lungs anteriorly. The right and left lungs weighed 1,013 g and 887 g respectively (NR 651 +/- 241 g, 579 +/- 221 g). On section there was marked congestion and expressible oedema. The cause of death was reported to be ischaemic heart disease with secondary drowning.

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹); carbon monoxide 6%.

Comments: It is likely that this man developed a cardiac arrhythmia or angina either during the dive or swimming on the surface, became disorientated and then unconscious, and subsequently drowned. It is unfortunate that his plight was not recognised and acted upon earlier as it is possible that he might have survived. However, it can be difficult for a lookout to assess the seriousness of such a situation. This case again highlights the importance of buddies staying together throughout a dive, and the potential, although rare, adverse consequences of allowing a diver to return to the boat or shore alone. Although the residual air in the cylinder tested met specifications, the blood carbon monoxide level was 6%. This would be consistent with a smoker, although

there is no information to confirm whether or not the victim had recently smoked.

Summary: Appeared healthy; no significant medical history declared; some experience; intentionally separated from buddies to return to boat; observed on surface by lookout but no distress evident; ‘silent’ unconsciousness; drowning (cardiac-related)

SC 09/05

This victim was a 50-year-old male who certified as a diver five years earlier and had logged approximately 75 dives, mostly shore dives in temperate waters. He also held Advanced Open Water and Nitrox certifications. The victim suffered from lumbo-sacral spondylosis as a result of a work injury and was being treated for hypertension and proteinuria, managed with telmisartan. He was taking rabeprazole sodium for reflux. He also suffered from idiopathic lymphoedema, being hospitalised on three occasions for cellulitis in his legs. He had given up smoking two years earlier and now went to the gymnasium about four times a week to help manage his spondylosis, swam regularly and appeared to be relatively fit although “*solid-looking and carrying a bit of fat*”. An ECG taken after an episode of chest pain a year earlier indicated “*normal tracing other than bradycardia*” (48 bpm). He had been certified fit to dive three months earlier by a doctor trained in dive medicine. This medical was sought as he was planning to upgrade his diving qualifications and was keen to eventually become an instructor.

He was now enrolled in a Rescue Diver course and was participating in some surface rescue drills. The group consisted of an instructor, an assistant instructor and four students. It was windy (gusts of up to 40 knots) but they dived from the shore in an area sheltered by a long breakwater and where the water was relatively calm. The victim was wearing mask, snorkel and fins, a semi-dry suit, hood, boots and gloves, BCD with 10.5 L cylinder, a regulator with ‘octopus’, dive computer and gauge and a weight belt with six weights (weight unreported). The water temperature was 12°C.

The group was in the water approximately 50 metres from shore and had been doing surface rescue tows for about 20 minutes. The depth was 2–3 msw. The victim had acted as both the rescuer (required to tow another diver about 30 metres), and the rescuee, which was the last role he had undertaken. He had not submerged at any time during the exercise. Shortly after completing a briefing, the instructors heard thrashing in the water and realised that it was from the victim, who was about 2 metres distant floating on his back. One of the other students turned to him and asked if he was “OK”, which he said he was. However, he then began thrashing and turning over in the water before becoming motionless, face-down. When one of the instructors went to him and rolled him over, he was unresponsive with

froth coming from his mouth, although he appeared to be breathing. While the instructors and another diver towed the victim towards the rocks he appeared to have a seizure and became apneic and cyanotic. One of the rescuers commenced mouth-to-mask rescue breathing as they towed the victim. Once they reached shore, the instructors began BLS while one of the students went to call an ambulance. When the ambulance arrived 16 minutes later, the paramedics initially had considerable trouble drying his chest sufficiently for the defibrillator pads to adhere. When this was achieved, the victim was found to be asystolic. An intravenous line was established and he was intubated and given 5 mg of adrenaline intravenously (5 x 1 mg). After about 15 minutes, a shockable rhythm appeared and he was given three shocks and transported to hospital where he was later pronounced dead.

When tested, his equipment was found to be functioning correctly, his cylinder was full, and the air met appropriate purity standards.

Autopsy: The heart weighed 498 g (NR 400 +/- 69 g) and had a normal external contour. There was an area of congestion in the posterior basal left ventricular wall. The left and right ventricular myocardium measured 16 mm (concentrically) and 6 mm in thickness, respectively. There was severe atherosclerosis with up to 80% stenosis of the LAD coronary artery, 90% stenosis of the first diagonal branch and 90% stenosis of the left circumflex coronary artery. The right coronary artery showed mild atheroma. The upper and lower airways were free of debris and foreign material. The lungs were congested. The right and left lungs weighed 914 g and 647 g respectively (NR 663 +/- 217 g, 569 +/- 221 g). The brain weighed 1,365 g (NR 1,423 +/- 161 g) and was normal. The right and left kidneys weighed 157 g and 141 g respectively (NR 169 +/- 37 g, 174 +/- 35 g) and appeared unremarkable macroscopically. Histology of the heart showed widespread vacuolation of the myocytes but no acute infarction or fibrosis. Renal histology showed occasional sclerosed glomeruli, a patchy interstitial lymphocytic infiltrate and slightly hypercellular intact glomeruli consistent with the history of previous renal disease. The cause of death was given as ischaemic heart disease (likely cardiac arrhythmia). **Toxicology:** alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: It is likely that the substantial exertion of the rescue exercises triggered an arrhythmia in this susceptible diver. What this case highlights is that, even those middle-aged divers who ostensibly have a good exercise tolerance and who exercise regularly can have occult severe coronary disease. The role of screening tests in divers of this age remains controversial because of the high false positive rates and complications of the invasive investigations.^{10,11} One hopes that new technologies such as MRI angiograms may enable non-invasive evaluation of the coronary vessels in individuals such as this. The rescuers acted swiftly and

appropriately, unfortunately to no avail.

Summary: History of spondylosis, hypertension, proteinuria, reflux, idiopathic lymphoedema and cellulitis; experienced diver; exercised regularly; recent dive medical; doing rescue training on surface; suffered seizure and became unconscious; cardiac death

SC 09/06

This 52-year-old male, a non-smoker and social drinker, was an active and competitive wood-chopper who was described as fit and healthy, although “*a bit overweight*”. His doctor regularly prescribed perindopril for hypertension, pravastin for hypercholesterolaemia, and naproxen periodically for musculoskeletal pains. Five months earlier, following an episode of what his doctor believed to be musculoskeletal-related chest pain, he had undertaken an exercise stress test. This concluded that “*the deceased displayed good exercise tolerance and no evidence of inducible ischaemia or symptoms at the prescribed workload.*” He had last seen his doctor two months earlier to monitor his hypertension and was reported to have been in good health.

He had been certified as a diver for eight years and had logged 65 dives, although he had not dived for over three years. His tank was tested several days before this incident. He had also recently bought a new mask and snorkel, which he had used the day before, complaining that he was unable to get a good mask seal and, although not flooding, the mask leaked just enough to be annoying. He and two friends were on a fishing trip and planned to scuba dive from one of the friend’s five-metre boat. On the day before the dive, the victim was reported to have drunk four beers and shared a bottle of wine and appeared to be well.

On this day, after abandoning fishing because of a strong wind, the group anchored the boat in the lee of a small island where the depth was around 4 msw. When assembling his gear, the victim initially tried attaching his regulator upside down before asking for help from one of his friends. The plan was for the victim to buddy up with one friend, an inexperienced diver, and to stay close together. The other would dive alone for the others’ safety, as he had a speargun. The wind was reported to be moderate (15 knots), the sea calm with no significant current or swell, water temperature 22°C and visibility 10–15 m.

The victim was wearing mask, snorkel and fins, a two-piece wetsuit, BCD, 11.5 L cylinder (filled to 250 bar), regulator, octopus and gauge, a knife and a weight belt (with an unconfirmed amount of weight, possibly 20 kg). He entered the water last and appeared to be relaxed underwater. However, on at least two occasions the buddy saw the victim kneeling on the seabed, clearing his mask, the last occasion being about 20 minutes into the dive. After his ‘OK’ signal was returned by the victim, the buddy swam off

to continue diving. However, after a few minutes, the buddy realised that he could not see the victim and commenced a cursory search before returning to the boat alone. On the way, he found the victim's mask and snorkel and became concerned when he surfaced and could not see the victim on the surface or in the boat, some 50–60 m away. He returned to the boat. His computer indicated that he had a dive time of 44 minutes. After a further 20 minutes, the other diver returned and radioed for assistance, to which two nearby vessels responded.

The crew on one vessel located the victim floating face-down on the surface approximately 200 m away from the dive boat. He was unconscious, his regulator was out of his mouth and he was not wearing his weight belt. He was brought aboard and his equipment removed. Breathing and pulse were noted to be absent. He was cyanotic and there was no discharge coming from his mouth or nose. Some rescue breaths were given and the victim was soon transferred to the other, larger vessel where there was oxygen equipment and an AED. The AED was attached but no shock was advised so the crew commenced BLS with supplemental oxygen until the vessel arrived in port approximately 45 minutes later. Waiting paramedics pronounced him dead.

When later examined by police, the equipment was found to be functioning in accordance with manufacturers' specifications and was maintained in good order. The victim's cylinder still contained air at a pressure of 170 bar and this air met most of the purity standards, although carbon dioxide (CO₂) was slightly higher than recommended (500 ppm – maximum allowable under AS2299.1:2007 is 480 ppm). His maximum depth indicator read 4.5 msw.

Autopsy: The heart weighed 510 g (NR 400 +/- 69 g) and showed concentric hypertrophy and dilatation of the left ventricle. The aortic valve was functionally bicuspid with fusion of two of the valve cusps. The trachea and bronchi were clear. The right and left lungs weighed 740 g and 618 g respectively (NR 663 +/- 217 g, 569 +/- 221 g). Both lungs were inflated and were plum-coloured and markedly congested. There was no description of intravascular gas at autopsy. The coronary arteries showed advanced complicated stenotic atheroma on histology. The cause of death was given as immersion in a man with aortic valvular disease.

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: The reviewing pathologist (CL) would describe this death as the result of the combined effects of ischaemic and valvular heart disease. The victim possibly became stressed by his leaking mask and this may have precipitated a cardiac event. It is likely that he surfaced and ditched his weight belt before losing consciousness. Unfortunately no one was available to help him because of buddy separation. In the absence of CT scan or description of gas present at autopsy it is impossible to evaluate the possibility of PBT/CAGE. It is unlikely that the slightly elevated carbon dioxide content in his air contributed to this death.

Summary: Hypertension, hypercholesterolaemia, episode of chest pain with negative stress test; bicuspid aortic valve, severe atherosclerosis and left ventricular hypertrophy; experienced but no diving for 3 years; leaking mask; separation; cardiac death

SC 09/07

This victim was a 53-year-old male. Despite a strong family history of heart disease, he appeared to be very fit and healthy and was not known to be taking any medications. It was reported that "*he avoided doctors*". A diver for 39 years, he had qualified as an instructor 20 years earlier and had logged over 2,000 dives. He was a paying passenger on a dive charter vessel on a day trip in sub-tropical waters. The 19-metre boat was carrying 13 passengers and eight crew. He did not declare any medical conditions to the operator prior to diving. The victim was wearing a mask, snorkel and fins, a wetsuit, BCD with integrated removable weights of 5.5 kg, and an 11.5 L cylinder with regulator and octopus, dive computer and pressure gauge. The sea was calm, water temperature 24°C with a current of approximately 0.5 knots. He was paired with an inexperienced buddy and they made an uneventful 40-minute drift dive to a maximum depth of 16.4 msw. After a surface interval of 85 minutes, they entered the water for another dive on a wreck sitting on the seabed at a depth of around 25 msw.

The victim led his buddy around the wreck for about 40 minutes, at which time the buddy indicated that he was low on air and wanted to surface, which they did. The buddy described the ascent as controlled and calm, but at a rate slightly faster than usual. They did not do a safety stop. They had ascended up the anchor line of another boat, and, after speaking with its skipper, the victim decided that he and the buddy should swim to their own boat, variously reported as 50–200 m away. Initially, the buddy was unable to swim unaided against the current so the victim towed the buddy for several minutes until the buddy was able to make headway unaided. After swimming what was likely around 100 m, the divers reached the mermaid line from the boat and began to pull themselves along it. However, when the buddy reached the boat he (and others on the boat) noticed that the victim was floating away, face-down and motionless.

One of the crew swam over to the victim and found him unconscious with his mask in place and with the regulator in his mouth. A tender came over to assist, the victim was dragged aboard and BLS was commenced. Once on the main vessel, BLS was continued, with supplemental oxygen provided during the 10- to 15-minute transit to a nearby resort. Once there, resuscitation efforts were continued by a paramedic, without success.

When tested later, the equipment was found to be functioning correctly. The cylinder still contained air at a pressure of 65 bar. However, the CO₂ content of the air was found to be higher than recommended (559 ppm), as was the water

content. The dive computer indicated that the victim had dived to a maximum depth of 24.6 msw with a dive time of 40 minutes.

Autopsy: A full body CT scan (performed 18 hours after death) showed extensive gas present in the left side of the heart extending through the arterial system to involve the aorta and its major branches both inferiorly and superiorly, in the carotid, vertebral and cerebral arteries, as well as the proximal coronary, subclavian and common iliac arteries, extending into the legs. There was a small amount of gas in the right side of the heart and frothy blood in the inferior vena cava. There was a large amount of gas in the liver and mesenteric vessels and in the thigh muscles, possibly in vessels. There was no pneumothorax. Gas was noted in the hip joint and eye. The brain weighed 1,410 g (NR 1,423 +/- 161 g) and showed numerous air bubbles within the surface vessels. Focal haemorrhage was noted in the petrous temporal bone. Gas was able to be aspirated from the aorta and left ventricle and atrium. The lungs were oedematous and the right and left lungs weighed 880 g and 760 g respectively (NR 651 +/- 241 g, 579 +/- 201 g). There was frothy fluid in the trachea. The heart weighed 360 g (NR 370 +/- 75 g). There was severe coronary atherosclerosis with 90% narrowing of the proximal LAD coronary artery and 95% narrowing of the right coronary artery. There was extensive old scarring of the posterior left ventricle. Histology shows focal haemorrhage in the atheromatous plaque in the right coronary artery and old infarction and eosinophilia suggesting early acute ischaemia in the myocardium without a neutrophilic infiltrate. The cause of death was given as arterial gas embolism with coronary atherosclerosis as contributory findings.

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹).

Comments: The interpretation of the finding of gas at 18 hours postmortem is complex. The gas in the liver and mesenteric vessels is probably decompositional and post-mortem decompression artefact, and the gas in the eye, hip and thighs is almost certainly post-mortem decompression artefact. There does appear to be more gas in the arterial system than in the venous system which could be interpreted as PBT/CAGE. However, in a diver dying of a cardiac arrhythmia due to ischaemic heart disease, one might expect that normal off-gassing of nitrogen via the lungs would be compromised and that post-mortem decompression artefact would therefore be increased. The gas may all be due to post-mortem decompression artifact; however, in the experience of the reviewing pathologist (CL), it is unusual to see so much in the arterial system and so little in the venous system but it is probably unreliable to try to interpret a CT scan done 18 hours after death. The reviewers believe that the cause of death was more likely to have been ischaemic heart disease than CAGE, given the description of the incident.

Although this highly experienced diver appeared to be fit and healthy, his substantial (presumably asymptomatic) cardiovascular disease may well have led to an arrhythmia,

triggered by the heavy exertion of swimming and towing his buddy against a current. Older divers, especially those with a known significant medical condition and/or relevant family history (such as this victim), are advised to have periodic health checks.¹¹ This victim, although thought to have been in good health, was reported to have “*avoided doctors*”. It is interesting to contemplate whether a medical review might have raised concerns, or whether his significant cardiovascular disease might have remained undetected as in some other cases in this series.

Summary: Strong family history of cardiovascular disease; apparently fit and healthy; highly experienced diver and instructor; exertion against current; cardiac death

SC 09/08

This 33-year-old male, overseas national currently living in Australia, was apparently reasonably fit, had no known health problems and was on no medications. He had undergone a diving medical one month prior to being certified as an Open Water Diver two months earlier. He had undertaken only three open water dives. His swimming competency was not reported.

On this occasion he went diving for crayfish with three friends, two of whom were also inexperienced divers. The victim was wearing a 5 mm one-piece wetsuit with boots and gloves, mask, snorkel and fins, BCD with integrated weights system and 2.7 kg of weights, a 15 L cylinder (filled to around 240 bar), regulator, demand valve and ‘octopus’ and a dive computer. He was carrying a camera.

They dived from rocks in a relatively calm bay, a site about which they had little knowledge, although one of the group had dived it before. Although their entry point was sheltered from the wind and looked calm from their vantage point, there was a strong wind (gusting up to 25 knots) and a two-metre swell and strong current on the other side of the rocks, some 100 metres distant, beyond a sheltered canal. The water temperature was about 22°C and visibility was 10 m. Following a thorough briefing including selecting an exit point, they geared-up on the rocks and entered the water.

After an underwater time of 33 minutes at a maximum depth of 17 msw, the divers surfaced. They had planned to do a five-minute safety stop but abandoned this after two minutes because of the strong surge. They surfaced on the seaward side of the rocks where the conditions were rough, with breaking waves and a strong current. Unable to access their pre-determined exit point, they swam towards the nearest rocks, reaching them about 20 minutes later. Two of the divers managed to scramble onto the rocks, while the victim and one friend struggled against the waves and current. The victim managed to climb onto the rocks but, while attempting to stand, was knocked over by a breaking wave and fell backwards into the water. He had swallowed some water and was coughing and struggling in the water and calling

for help. One of the divers told him to put his regulator back in his mouth, which he did, before disappearing under the breaking waves without his mask on. A diver tried to jump in to help but was smashed against the rocks by the waves and retreated, losing his mask and fins in the process. Some of the others also lost equipment, and one sustained a fractured finger. The divers waited for a short time in the hope that the victim might surface away from the waves. However, when this seemed unlikely, they called the police.

Police arrived about 40 minutes after the victim was last seen and began a search, utilizing boats and a helicopter. The victim's body was seen from the surface by one of the searchers, close to where he had last been seen. He was lying face-up at a depth of 11 msw; his mask was missing and his regulator out of his mouth but his other equipment was still in place. When the searchers dropped a weighted line to mark the spot, it wrapped around the victim's arm enabling them to pull him to the surface. No attempts were made to resuscitate him.

When examined later by police, the equipment, which looked near-new, was found to be functioning in accordance with the manufacturers' specifications. The cylinder valve was fully closed which caused some initial concern. However, this was thought likely to have been done inadvertently by one of the searchers, although none admitted to doing so. When tested, the air met most of the required standards. The air pressure was 80 bar. However, the CO₂ content (500 ppm) was slightly higher than recommended, and the water content, at 160 mg m⁻³, was well above the recommended 50 mg m⁻³ (although this was not believed to have been contributory).

Autopsy: The post-mortem examination and CT scan were performed five days after death. A post-mortem CT scan showed fluid within the facial sinuses. There was no evident intracranial, intravascular or other compartment gas or evident trauma to the head or neck area. There was a column of fluid filling the trachea from the sternal notch to the carina. There were superficial bruising and abrasions on the face, arms and legs. There were petechiae on the conjunctivae. The heart weighed 266 g (NR 342 +/- 58 g) and was normal, with no coronary atherosclerosis. The lungs showed bilateral pulmonary oedema. The right and left lungs weighed 472 g and 448 g respectively (NR 670 +/- 249 g, 593 +/- 224 g). There was a small amount of frothy fluid in the upper airways. However, there was 600 ml of pale red fluid in the right pleural cavity, 500 ml in the left pleural cavity and 278 g watery fluid in the stomach. There were bilateral middle ear haemorrhages. The lung changes of drowning were obscured by decomposition; a prolonged post-mortem interval makes diagnosis harder, but some of the fluid appears to have moved into the pleural cavity. The cause of death was given as drowning.

Toxicology: alcohol detected 0.01% (probably from decomposition).

Comments: Through insufficient local knowledge and inexperience, these divers grossly underestimated the sea conditions that they would encounter when diving beyond the sheltered area where they entered. Had the victim inflated his BCD and ditched his weight belt, he might have survived. His inexperience and/or incapacitation from being swept back into the sea and aspirating and swallowing water likely hampered his ability to deal with these circumstances. Some divers find it difficult to breathe from a regulator underwater without a mask in the best of circumstances, much less under such adverse conditions. This is a skill that all divers should refresh periodically.

Summary: Healthy; recently certified and inexperienced; surfaced into rough conditions; swept from rocks; drowning

Surface-supply breathing apparatus (SSBA) diving fatalities (Table 2)

SS 09/01

This victim was a 36-year-old male who was described as fit and healthy. He had a history of bipolar affective disorder, although he was reported to have been happy and in good spirits at the time prior to this accident. Two years earlier, he had suffered what had been reported as a possible seizure, but there had been no known subsequent events. He had been a recreational diver for 13 years but his experience was unreported. He was assessed as fit to dive by a qualified diving medical examiner five months earlier when he began working at a pearl farm.

On this day, he was tasked to spread some pearl shell panels across the ocean floor and was diving from a tender. There were two other divers in the water doing different tasks, and a supervisor remained on board the tender, which was tethered to a platform. The victim was wearing a mask, fins, a two-piece wetsuit with undergarments, a weight belt (weight unreported) and was using a tow-line SSBA. He had positioned one set of panels about 30–40 m away and was returning towards the tender at a depth of 8 msw when, after a bottom time of 8 minutes, he ascended directly to the surface (rate approximately 18 m min⁻¹, indicated by his dive computer). On surfacing, he ditched his weight belt and was seen to wave for help before sinking to the bottom. The supervisor recalled the other divers and pulled the victim onto the platform by the airline. His mask was removed and there were froth and stomach contents coming from his mouth.

BLS was begun by the others and supplemental oxygen was added when the equipment was brought to the vessel. Resuscitation attempts continued for an hour, in consultation with a doctor by phone, until that doctor advised that efforts should be abandoned.

When tested, the equipment was found to be functioning adequately. There was a slightly elevated CO₂ content (500 ppm) in the air in his cylinder. A subsequent WHS inspection of the operation found some non-compliance issues but nothing that was believed to have contributed to this death.

Autopsy: The heart weighed 420 g (NR 400 +/- 69 g) and was reported to have appeared to be enlarged. The left ventricular wall measured 15 mm (NR < 14 mm). The coronary arteries showed minimal atheroma. The upper airways contained small quantities of frothy mucoid fluid. The lungs showed oedema and congestion. The right and left lungs weighed 850 g and 790 g respectively (NR 663 +/- 217 g, 569 +/- 221 g). There were petechiae on the conjunctivae. The brain weighed 1,640 g (NR 1,423 +/- 161 g). The cause of death was given as drowning.

Toxicology: alcohol undetected (< 10 mg 100 mL⁻¹); carbon monoxide 5% (consistent with a chronic smoker).

Comments: The pathologist suggested that the heart was enlarged and that cardiac causes could have contributed to the drowning. However, the heart weight is within the normal range for his BMI, and the reviewing pathologist (CL) has questioned whether the heart was in fact enlarged. Although it is unknown what caused this diver to ascend and call for help, several possible factors could have precipitated unconsciousness and subsequent drowning. These include a cardiac arrhythmia (as suggested by the examining pathologist), which could have resulted from exertion underwater; or a PBT/CAGE. As there was no imaging of the body and no description of gas found at autopsy, it was not possible to evaluate PBT/CAGE on this autopsy.

Given his history of what was thought to have been a seizure, it is possible that he had another seizure and subsequently drowned. It is also possible that he simply aspirated water inadvertently, although this seems to have been unlikely. It appears that about eight minutes elapsed from the time he surfaced and waved for help to when he was dragged onto the platform. Although longer than ideal, this indicates a relatively rapid rescue under the circumstances and one can speculate whether or not an AED might have been beneficial in this case.

Other than a comment by a family member, no information was available about the possible seizure suffered by the victim several years earlier. It is probable that this information was withheld from the diving medical examiner as, otherwise, it is unlikely that the victim would have been given a medical clearance to dive. Unfortunately our research team was unable to view all of the relevant documents for this case so some pertinent information might have been missed.

Summary: Apparently fit and healthy although history of seizure and bipolar affective disorder; certified for 13 years but experience unknown; recent dive medical; diving alone

on commercial pearl farm; some exertion; surfaced and called for help; ditched weights but sank; drowning

Root cause analysis

The root cause analysis for each case reported is summarized in Table 3.

Discussion

APNEIC HYPOXIA

It seems likely that three of the victims in this series (BH 09/07, BH 09/09 and BH 09/10) died as a result of apneic hypoxia (often referred to, incorrectly, by the lay community as “*shallow water blackout*” but, perhaps, as suggested by one author (JL), better described as “*breath-holding blackout*”). All of these victims were young and apparently fit and healthy, which is consistent with many previous cases.¹²⁻¹⁶ Pushing one’s breath-holding limits is a potentially dangerous practice and sudden unconsciousness can occur with or without hyperventilation. Ascent from depth increases the likelihood of this as a result of the rapid reduction in ambient pressure and consequent drop in partial pressure of oxygen in the blood and tissues. However, as is likely with BH 09/07, sudden unconsciousness can occur as a result of extended breath-holding with minimal ascent. DAN AP Australian data indicate that, between 2004 and 2009, four snorkellers died while practising extended apnea in a swimming pool.¹⁷ In the absence of a readily available rescuer this practice can carry unacceptably high risks.

BUDDY SYSTEM / SUPERVISION

A recurring theme in dive fatality reports is the absence, or breakdown, of the ‘buddy system’.¹²⁻¹⁹ This series is no exception. Only seven of the 21 victims (33%) were with their buddy or under close supervision when the accident occurred. Four of the victims had dived solo, five had intentionally separated from their companions during the dive, three were under indirect supervision, and two victims had accidentally separated from their buddies. It is important to remember that an unconscious, apneic diver needs to be rescued and resuscitated swiftly in order to have a chance of survival. This is extremely unlikely in the absence of a vigilant buddy or close supervision, and sometimes impossible even if present.

In commercial snorkelling situations there are often too few lookouts relative to the number of snorkellers and this makes adequate supervision difficult. A snorkeller who appears to be fine one minute can suddenly and silently become unconscious soon after, and several minutes may elapse before this is recognised by a lookout who is required to monitor what often becomes a dispersed group. The use of relatively few lookouts might be valid in ideal conditions. However, increased vigilance (usually including more lookouts) is necessary where conditions are less

Table 3
Root cause analysis of diving-related fatalities in Australian waters in 2009

Case	Trigger	Disabling agent	Disabling injury	Cause of death
BH 09/01	Exertion?	Ischaemic heart disease	Cardiac incident	Drowning
BH 09/02	Exertion? Immersion?	Ischaemic heart disease	Cardiac incident	Cardiac-related
BH 09/03	Strong current	Buoyancy-related	Asphyxia	Drowning
BH 09/04	Unknown	Ankylosing spondylitis?	Asphyxia? Cardiac incident?	Drowning
BH 09/05	Exertion	Cardiovascular disease	Cardiac incident	Cardiac-related
BH 09/06	Immersion? Aspiration?	Cardiovascular disease?	Cardiac incident?	Drowning
BH 09/07	Prolonged breath-holding	Apneic hypoxia? Cardiac (fibro-muscular dysplasia)?	Asphyxia? Cardiac incident?	Drowning
BH 09/08	Inexperience/panic? Postural effect on brainstem?	Aspiration? Sudden unconsciousness?	Asphyxia	Drowning
BH 09/09	Prolonged breath-holding? Dragged under by shark?	Apneic hypoxia? Buoyancy-related?	Asphyxia	Drowning
BH 09/10	Prolonged breath-holding	Apneic hypoxia	Asphyxia	Drowning
BH 09/11	Feeling unwell? Current?	Aspiration	Asphyxia	Drowning
BH 09/12	Immersion? Aspiration? Asthma	Aspiration? Cardiac disease?	Asphyxia? Cardiac incident?	Drowning
SC 09/01	Epilepsy? Rough water?	Unconsciousness from seizure? Impact with rocks?	Asphyxia	Drowning
SC 09/02	Unknown	Aspiration? Rapid ascent?	Asphyxia? CAGE?	Drowning
SC 09/03	Unknown	Unknown	Unknown	Unknown
SC 09/04	Immersion? Exertion?	Cardiovascular disease	Cardiac incident	Drowning
SC 09/05	Exertion	Cardiovascular disease	Cardiac incident	Cardiac-related
SC 09/06	Leaking mask?	Cardiovascular disease	Cardiac incident	Cardiac-related
SC 09/07	Exertion	Cardiovascular disease? Ascent-related?	Cardiac incident? CAGE?	Cardiac-related? CAGE?
SC 09/08	Rough water	Aspiration	Asphyxia	Drowning
SS09/01	Exertion?	Cardiovascular disease? Epilepsy? Ascent-related?	Cardiac incident? CAGE? Seizure?	Drowning

than optimal, and rigor is required when deciding whether conditions are suitable for novice snorkellers.

Tragically, one of the victims (SC 09/03) was doing an introductory scuba programme under the supervision of an instructor in circumstances where it was arguable that the instructor-student ratio was inappropriate.

EPILEPSY

Although diving with epilepsy is still the subject of ongoing debate,⁸ the South Pacific Underwater Medical

Society recommends that “a candidate with a history of fits (apart from childhood febrile convulsions), or unexplained blackouts should be strongly advised against diving. Any condition associated with fits or blackouts will be a grave risk to life during diving.”²⁰ Diving and/or snorkelling may involve several factors that can reduce the seizure threshold, such as stress, exercise, sensory deprivation, hypercapnea, hyperventilation and hypothermia. Seizures can occur while taking anti-epileptic medication or may recur many years after the cessation of medication.⁹ Two of the victims in this series had a reported history of epilepsy (SC 09/01, SS 09/01). Given that there were no witnesses to these accidents

(both divers were diving solo), and the inability to detect medical evidence of a seizure at autopsy, it is impossible to confirm whether or not a seizure contributed to these deaths. However, these authors believe people with a history of or current epilepsy are well advised to refrain from scuba diving and to snorkel only if closely supervised.

GAINING POSITIVE BUOYANCY IN AN EMERGENCY

It was necessary to conduct an underwater search to locate the bodies of three of the divers in this series (SC 09/02, SC 09/03 and SC 09/08). The need to locate a diver, especially underwater, prevents early resuscitation efforts and it is far better for an unconscious diver to be at the surface, rather than to have to be searched for and recovered from depth. This is evidenced by a Canadian report of the outcomes of 37 scuba diving accidents.²¹ Of 13 divers who failed to surface and for whom a search was required, 12 died and only one survived. Of the 25 divers who managed to reach the surface after getting into difficulties, 22 survived, despite some requiring resuscitation and/or recompression.²¹ Sometimes a stricken diver simply does not have the opportunity to gain positive buoyancy by inflating his BCD and/or ditching his weight belt or integrated weights. However, this is an important educational message and training drill, which needs to be practised, embedded and periodically re-visited.

OVERWEIGHTING OF TRAINEE DIVERS

In SC 09/03, the student appears to have been over-weighted. Divers are advised that they should adjust their weights so that they are 'neutrally buoyant' on the surface (or at their safety stop). Despite this, it is common practice for instructors to make their students negatively buoyant as it usually makes them less likely to inadvertently float to the surface during a dive without the instructor's knowledge. Although this is not ideal, it generally achieves the stated goal. An additional factor is that many novice divers breathe at a larger lung-volume range than in normal, relaxed breathing, thus increasing their positive buoyancy (body buoyancy being most dependent on that created by the gases in the lungs). As they learn to relax with increasing experience, this changes and a concomitant reduction in weighting is needed, but is often not made. Unless the student or instructor adjusts the student's buoyancy properly using their BCD (students are purposely not trained to do so while underwater in an introductory dive), they tend to swim in a semi-upright position, which increases resistance through the water, or drag themselves along the bottom, so stirring up sediment, damaging reef and reducing visibility. This has the potential to cause fatigue. Additionally, and of important relevance to SC 09/03, over-weighting can make it very difficult for the diver to ascend and remain on the surface unless they sufficiently inflate their BCD and/or ditch their weight belt. The importance of the relationship of lung volume to correct buoyancy is generally not well explained.

CARDIAC-RELATED DEATHS

In this series it is again apparent that cardiac-related factors represent a major disabling injury in both snorkelling and compressed-gas diving incidents. These appear to have contributed to at least six, and possibly up to 12 of the 21 fatalities for this year. Australian fatality data from 2004 to 2009 inclusive indicate that, between 34 to 46 of the 120 diving-related fatalities (28–38%) appear to have been caused by a cardiac incident.¹⁷ This is reasonably consistent with DAN America data from 1992 to 2003 which yielded a rate of 26.4%.¹² However, it is far higher than the 18% of apparent cardiac-related deaths reported for Australia between 1972 and 2005.¹⁸ The difference is undoubtedly partly owing to the increasing participation of older divers, with the likely associated occult cardiac disease. It is also likely owing in part to the increasing awareness of examining forensic pathologists of the potential impact of diving on a person with cardiac disease.

Increasing age and obesity are known indicators of increased risk of a cardiac incident.^{22,23} In this series, the ages of the six victims who likely died from a cardiac cause ranged from 50 to 63 years. Their BMIs ranged from 24.2 to 32.3 kg m⁻¹. One victim who was classified as a possible cardiac-related death was morbidly obese, with a BMI of 42 kg m⁻². A poor swimmer, she failed to declare various medical conditions and associated medications and was 'an accident waiting to happen'. A snorkel operator is faced with a conundrum when dealing with such a customer. While the declaration of a medical condition may provide an avenue of refusal, one must be careful not to be discriminative on the basis of obvious obesity. If there is pressure from the individual to be allowed to participate, which is often the case, then the ideal situation is to allocate a guide to accompany the person, possibly on a one-to-one or one-to-two basis to enable the provision of rapid response and support if required. However, staff numbers often do not enable this, or the individual may not agree to pay any extra associated costs. In such circumstance, dive operators need to show the courage to decline such individuals on safety grounds based on their risk assessment.

The issue of medical screening of the ageing diver is a large subject in its own right. The likelihood of occult coronary artery disease increases with age, and this seems to be reflected in these mortality reports. The difficulty facing the diving medical examiner is selecting which diver candidates to investigate further, and what are the most appropriate

* **Footnote 2:** DAN Asia-Pacific has recently launched an awareness campaign in an attempt to better educate the diving community of the potential danger of diving with cardiac disease. A poster can be downloaded at: <http://www.danasiapacific.org/main/_images/DAN_Cardiac_Poster.jpg>.

investigations to choose. All investigations have a cost and an associated risk, and are prone to false positive and false negative results. This matter represents a current conundrum in diving medicine.

INTRODUCTORY DIVE RATIOS

Training agencies have standards to which their instructors are obliged to adhere, and these include guidelines on training, instructor-to-student ratios, equipment and various other factors. Ratios are set for ideal conditions and should be reduced accordingly if conditions are less than ideal. Despite the general safety of diver training, unforeseen circumstances, medical factors, human error, carelessness, ignorance and occasional negligence may intervene and a student come to serious harm. In an extensive 2010 paper based on PADI worldwide training data, it was reported that between 1999 to 2008 there were 24 recorded deaths in a total of 2,104,366 reported Discover Scuba Dives (i.e., introductory dives); a death rate of 1.14 per 100,000 dives.²⁴

It is questionable whether introductory dive programmes should be conducted at all in poor environmental conditions, based on consideration of participant pleasure with the consequent future potential to take up diving and on safety grounds. From a safety perspective, with an introductory dive such as the one reported here as SC 09/03, it may be more sensible for an instructor (and their employer, if relevant) to consider a ratio starting point of one or two students, and work upwards if the conditions are considered suitable, rather than starting from the maximum and working down. The mindset is subtly different and might help to err on the side of safety. However, this suggestion is likely to be unpopular commercially.

CARBON DIOXIDE CONTENT OF BREATHING GAS

In four fatalities (SC 09/06, SC 09/07, SC 09/08, SS 09/01), the analysis of the breathing gas demonstrated carbon dioxide levels of approximately 500 ppm, slightly in excess of the current Australian Standard (AS2299.1) of 480ppm. In each case, this was thought to be non-contributory to the fatality. It is interesting to note that the current standard for the Royal Australian Navy (RAN) is 800 ppm and for the US Navy, 1000 ppm. Furthermore, AS2299.1 is presently under review, and it is likely that the new standard will allow for a higher level of CO₂ (Ian Millar, personal communication, 2013). A CO₂ of 500 ppm does not necessarily reflect air-supply contamination as the CO₂ in the 'urban dome' (inner city) can often exceed 550 ppm, depending on the time of year and location.²⁵

The American Conference of Industrial Hygienists allows for a continuous exposure of 5,000 ppm (i.e., USN standard @ 40 msw) with a short-term exposure limit of 30,000 ppm (50,000 ppm produces intoxication and greater concentrations may produce unconsciousness). Thus, the

present AS 2299.1 is very conservative even allowing for the effects of pressurisation. In all likelihood, the levels seen in these gas samples were the result of environmental CO₂. Whilst the slightly elevated carbon dioxide and water vapour in several cases was non-contributory, it is a timely reminder that air compressors must be meticulously maintained, and commercial operations have a testing and certification obligation.

Limitations of the study

As with any uncontrolled case series, there were inevitable limitations and uncertainties associated with our investigations. These included:

- Incomplete case data. Fatalities were sometimes unwitnessed, and reports provided by any witnesses and by police varied in their likely reliability, as well as the content and expertise of the investigators.
- Autopsy reports can sometimes be unreliable as a result of the difficulty of determining the presence of CAGE in the absence of relatively prompt post-mortem CT scans, and the inability to detect evidence of cardiac arrhythmias, among other factors. Care must be taken to critically examine the available evidence and minimise speculation when determining the likely disabling injuries.
- Classification of cases into a sequence of four events (trigger, disabling agent, disabling injury, cause of death) using root cause analysis (Table 3) requires a single choice for each component event, which may omit important factors in some cases because, at each level, multiple factors rather than a single one may be at play.²
- Limited annual case data. Twenty-one deaths are too few to reliably determine trends.

Conclusions

In 2009, there were 21 reported fatalities, including 18 males and three females. Twelve deaths occurred while snorkelling and/or breath-hold diving, eight while scuba diving, and one using SSBA. Apneic hypoxia continues to be a problem with breath-hold divers and appears to have caused the demise of three victims. Cardiac-related issues, particularly in older divers, were thought to have been the disabling injury in the deaths of at least three snorkel divers and at least three of scuba divers and may have been contributory in several others. One of the victims in this series was a student who became separated from her instructor on an introductory scuba dive in poor visibility. Other causal factors associated with these deaths that may have been contributory included inexperience, non-existent or poor buddy systems, inadequate supervision and diving in rough conditions.

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John Lippmann is Executive Director of DAN AP. DAN is involved in the collection and reporting of dive accident data and provides evacuation cover and dive injury insurance to recreational divers. This study is funded by DAN AP.

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Effects of hyperbaric oxygen on blood glucose levels in patients with diabetes mellitus, stroke or traumatic brain injury and healthy volunteers: a prospective, crossover, controlled trial

Ronit Koren Peleg, Gregori Fishlev, Yair Bechor, Jacob Bergan, Mony Friedman, Shlomit Koren, Amit Tirosh and Shai Efrati

Abstract

(Peleg RK, Fishlev G, Bechor Y, Bergan J, Friedman M, Koren S, Tirosh A, Efrati S. Effects of hyperbaric oxygen therapy on blood glucose levels in patients with diabetes mellitus, stroke or traumatic brain injury and healthy volunteers: a prospective, crossover, controlled trial. *Diving and Hyperbaric Medicine*. 2013 December;43(4):218-221.)

Introduction: A decrease in blood glucose levels (BGL) during hyperbaric oxygen treatment (HBOT) is a well-recognised phenomenon, but studies of this are limited and inconclusive. This study evaluated the effect of HBOT on BGL in patients with diabetes mellitus (DM), traumatic brain injury (TBI) or stroke and healthy volunteers in a prospective, open, controlled trial.

Methods: Thirty-nine participants were enrolled and evaluated twice: once during HBOT (90 minutes at 203 kPa), and once during a control session on normobaric air. Sessions were held up to two weeks apart and participants were instructed to eat the same diet. BGL was measured before, during and at the completion of each session.

Results: For the whole study group, there was a small but statistically significant decrease in BGL in both the HBOT (7.27 ± 3.66 mmol L⁻¹ before to 6.71 ± 3.88 mmol L⁻¹ after, $P = 0.037$) and control (air) sessions (7.43 ± 3.49 mmol L⁻¹ before to 6.71 ± 3.77 mmol L⁻¹ after, $P = 0.004$). This fall did not differ between the two conditions ($P = 0.59$). Examining the three groups separately, BGL fell in all three subgroups, but this fall was only statistically significant for the air session in the diabetic group. There were no statistically significant differences in the BGL reduction when HBOT was compared to normobaric air in any of the three subgroups.

Conclusions: BGL may decrease during HBOT and accordingly it should be monitored before entering the chamber. However, this decrease in BGL should probably not be attributed to the hyperbaric environment per se.

Key words

Hyperbaric oxygen, blood glucose, prospective controlled trial, blood pressure, heart rate

Introduction

Hyperbaric oxygen treatment (HBOT) is an established treatment for a variety of acute and elective medical conditions and usually involves exposure to 100% oxygen at a pressure of 152–284 kiloPascal (kPa).^{1–3} The main purpose of HBOT is to facilitate the repair of damaged hypoxic tissues. Accordingly, there is an increasing use of HBOT for relatively high-risk groups of patients, such as those with diabetes mellitus (DM) and its related complications or patients with neurological deficits resulting from brain injury (post radiation, stroke or traumatic brain injury, TBI). These populations are relatively vulnerable and more prone to be affected by changes in their blood glucose level (BGL) as well as in haemodynamic parameters.

In a study comparing BGL in five diabetic patients with five healthy volunteers treated with HBOT at 203 kPa for 90 minutes, a decrease in BGL was seen in the diabetic group but not among volunteers.⁴ Another study investigated the influence of HBOT on patients with and without DM and hypertension (HTN).⁵ Patients were exposed to 100% O₂ at 203–254 kPa in a monoplace chamber for 60–90 minutes. BGL decreased after treatment in the DM group. In a controlled study of 27 patients with DM, 13 insulin-dependent, eight on oral hypoglycaemics and six controlled on diet alone, a decrease in BGL was found after HBOT.⁶

This was most evident in the insulin-dependent patients. A comparison was also made with normobaric air for five of the patients, with no similar decrease of BGL.⁶ The major limitation for these clinical studies was the lack of a control group or a relatively small study group.

Therefore, the effect of HBOT on BGL in patients with DM and volunteers is inconclusive and the effect in patients with neurological deficits, who are more prone to convulsions, is unknown. The primary objective of the current study was to evaluate the effect of HBOT on BGL in patients with DM, brain injury due to stroke or TBI and healthy volunteers in a prospective, crossover, controlled trial.

Methods

STUDY POPULATION

Subjects were recruited from patients treated in the Hyperbaric Institute of Assaf Harofeh Medical Center, Israel. Participants were patients with Type 2 DM, insulin-(IDDM) and non-insulin-dependent (NIDDM), treated for non-healing wounds, or patients with TBI or stroke treated for neurological deficit. Healthy volunteers were recruited as well. Exclusion criteria were: patients who refused or could not sign an informed consent; recent ear surgery or ear problems; claustrophobia; chest X-ray pathology;

Table 1
Patient characteristics; mean (standard deviation); BMI – body mass index

	All subjects (<i>n</i> = 39)	Healthy volunteers (<i>n</i> = 13)	Diabetic patients (<i>n</i> = 13)	Neurological patients (<i>n</i> = 13)
Age (years)	51 (17)	32 (7)	74 (7)	57 (14)
Sex (M/F)	16/23	5/8	3/10	8/5
Smokers	8	1	5	2
BMI (kg m ⁻²)	26 (6)	22 (3)	31 (7)	25 (4)
Hypertension	12	0	8	4

chronic lung disease, sinusitis or respiratory tract infection and pregnancy. The protocol was approved by the Assaf Harofeh Medical Center Helsinki Committee for Human Experiments, approval number 218/10.

STUDY DESIGN

Patients were recruited after at least 10 HBOT sessions in order to reduce confounders such as anxiety, and to allow adjustment to the chamber environment. At baseline, all patients underwent a physical examination and a chest X-ray. Patients were evaluated in two separate sessions. In the first session, participants underwent full HBOT for 90 minutes at 203 kPa. On a different day, with a time interval of between one and 14 days, the control session, with room air at sea level pressure, took place in the hyperbaric unit and lasted for the same duration as the HBOT exposure. The two sessions were held at the same time of day. In order to disrupt patient care as little as possible and to enhance compliance with the study, all patients had HBOT prior to undertaking the control session. Participants were instructed to eat the same meal of 300 calories (including 50 grams of carbohydrates) two hours prior to each of the two sessions and to continue all medications including oral hypoglycaemics and insulin as usual. Whole blood was drawn for the measurements of BGL before, in the middle of and at the end of each session. Glucose was measured by an enzymatic colorimetric assay with a Roche/Hitachi 912 analyzer. Arterial blood pressure, heart rate and oral temperature were monitored during the study.

STATISTICAL ANALYSIS

BGL is expressed as mean \pm standard deviation (mmol L⁻¹) and within-subject changes were compared using Student's paired *t*-tests. The two sessions (HBOT and air control) were compared by repeated measures ANOVA test. Non-parametric data are expressed as absolute numbers. The before and after data are presented.

Results

Forty-two participants signed informed consent; three withdrew consent prior to the initiation of the study. Thirty-nine participants were included in the final analysis, 13 patients with DM, 13 patients with stroke or TBI and 13 healthy volunteers. Demographic and clinical characteristics

are presented in Table 1. In the diabetic group eight were controlled with oral hypoglycaemics and five with insulin. The mean interval between the HBOT and the control sessions was 6 days (range: 1–14 days). There were no differences in the baseline physiologic parameters measured at the beginning of the control and HBOT sessions.

BLOOD GLUCOSE LEVELS

For the whole study cohort, there was a small but statistically significant decrease in BGL in both the HBOT (7.27 ± 3.66 mmol L⁻¹ before to 6.71 ± 3.88 mmol L⁻¹ after, $P = 0.037$) and control (room air) sessions (7.43 ± 3.49 mmol L⁻¹ before to 6.71 ± 3.77 mmol L⁻¹ after, $P = 0.004$), but this fall did not differ between the two sessions ($P = 0.59$). Examining the three groups separately, BGL fell from baseline in all three groups, but this fall was only statistically significant during the control (room air) session in patients with DM (Table 2). There were no statistically significant differences in the BGL reduction when HBOT was compared to the control air session in each of the three study subgroups.

The insulin-dependent patients had no change in BGL either during HBOT (13.0 ± 4.0 mmol L⁻¹ before to 13.2 ± 5.7 mmol L⁻¹ after, $P = 0.88$) or during the control session (13.15 ± 2.7 before to 13.2 ± 4.7 mmol L⁻¹ after, $P = 0.96$). The NIDDM patients had a significant decrease in their BGL during both sessions; from 9.2 ± 3.0 mmol L⁻¹ to 7.3 ± 3.0 mmol L⁻¹ ($P = 0.047$) during HBOT and from 9.9 ± 2.9 to 7.8 ± 3.4 mmol L⁻¹ ($P = 0.004$) during the control session.

Since there were clearly no significant differences between the groups, the study was terminated after a total of 39 patients had completed the protocol. Post-hoc power analysis gave 83.8% power, $\alpha = 0.2$, for detection of a 30% difference in BGL between the groups for $n = 13$ in each group.

Discussion

Many patients treated electively with HBOT suffer from DM or neurological deficits due to stroke or TBI. These populations are relatively susceptible to fluctuations in BGL. It is well known that diabetic patients are at increased risk for hypoglycaemic events during HBOT. However, there might be other factors unrelated to the hyperbaric environment responsible for the fluctuation in BGL, such

Table 2
Effect of HBOT on blood glucose levels and comparison to control session on room air; mean (standard deviation);
HBOT – hyperbaric oxygen treatment

Group	Session	Baseline	End of session	P value
All subjects	Room air	7.43 (3.49)	6.71 (3.77)	0.004
	HBOT	7.27 (3.66)	6.71 (3.88)	0.037
Diabetes group	Room air	11.15 (3.38)	9.82 (5.0)	0.036
	HBOT	10.71 (4.05)	9.6 (5.32)	0.15
Neurologic group	Room air	6.38 (1.83)	5.7 (1.6)	0.08
	HBOT	6.38 (2.21)	6.0 (2.1)	0.1
Healthy group	Room air	4.71 (0.5)	4.5 (0.4)	0.37
	HBOT	4.71 (0.38)	4.55 (0.27)	0.25

as diet (for example, fasting during transportation and waiting time), maladjustment of glucose lowering drugs, decrease in catecholamine levels and changes in BGL and insulin requirements during infectious or other intercurrent diseases.⁷

Previous studies in humans, summarised earlier, were relatively small, uncontrolled or included mainly insulin-dependent diabetics.⁴⁻⁶ For example, the study by Ekanayake et al did show a decrease in BGL compared with a control session but it included a relatively small cohort of patients, mostly insulin-dependent diabetics.⁴ In the study by Trytko et al, although 27 patients were studied during a total of 237 sessions, a control session using room air was held in only five of them.⁶ They found that a decrease in BGL of more than 2 mmol L⁻¹ was associated with HBOT. Since there was a wide variability in BGL measurements, it is not possible to know which patient on which session had a significant decrease in BGL. Trytko's study included 14 non-insulin-dependent diabetics (six of them controlled with diet alone) and 13 were insulin-dependent; the more prominent decrease in BGL occurred in the insulin-dependent patients. In our study, in the patients with DM, there was a decrease in BGL only in NIDDM patients but this decrease was not related to the hyperbaric environment.

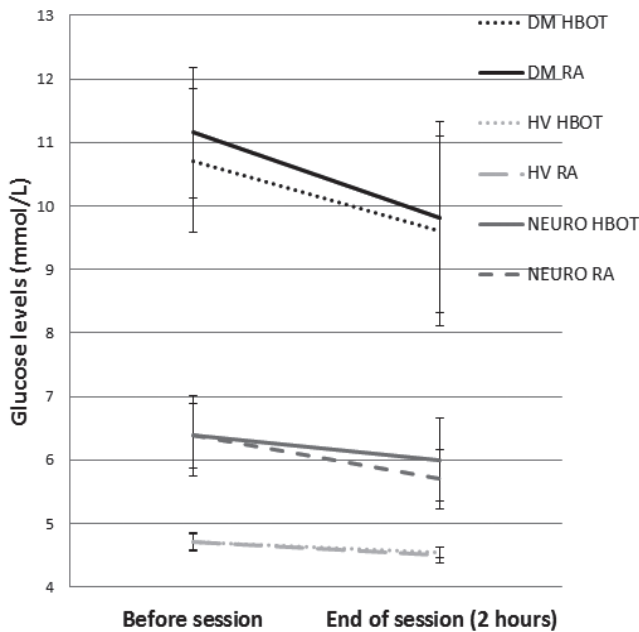
This study aimed to evaluate whether the change in BGL is related to the hyperbaric oxygen environment per se. The question was investigated in a prospective, crossover, controlled manner. The study cohort was chosen to be representative of the typical population of patients treated electively with HBOT. Type II DM patients suffering from non-healing ulcers represent one of the largest populations treated by HBOT, whilst TBI and post-stroke patients are a relatively new group of patients being studied and treated with HBOT. Recently, HBOT has been shown to induce neuroplasticity and improve brain metabolism in post-stroke patients.⁸ Stroke patients are more prone to seizures, a tendency that rises with stroke severity.⁹ Brain metabolism is highly glucose-dependent and, under normal conditions, the brain utilizes 25–30% of the overall body glucose consumption. As demonstrated in rat models,

HBOT increases brain glucose utilization and accordingly increases its susceptibility to any reduction of blood glucose concentration.^{10,11} HBOT may also increase insulin secretion in diabetic patients, in addition to improving insulin resistance.^{12,13} Currently there is no literature concerning the effect of HBOT on blood glucose in TBI or post-stroke patients. However, since brain injury, in addition to change in glucose utilization, can lead to autonomic and/or endocrine changes, it is important to explore whether this group would have a different response to HBOT. Another important reason for including this group of patients is their relatively high risk for seizures. Since hypoglycaemia by itself can cause seizures and patients with brain injury are more prone to seizures, it is important to investigate this issue.

The results indicate that the decrease in BGL was similar during HBOT and during a normobaric air session in all three subgroups. This suggests that food deprivation during the treatment and the control sessions, and not the hyperbaric environment, is probably the primary cause for the decrease in BGL. In the diabetic subgroup, there was no significant decrease in BGL during HBOT, which is also true for the subgroup of insulin-dependent patients. Unlike previous studies, the BGL at the beginning of each session was two hours postprandial. This can serve as a possible explanation for the absence of hypoglycaemic episodes. Since each patient had the same meal with the same drug regimen, and since the evaluation sessions were held at the same time of the day, it can be assumed that the hyperbaric effect on BGL was isolated. Furthermore, the same effect on BGL was apparent among healthy volunteers and post-stroke and TBI patients. Another possible explanation for the lack of difference may be a study effect: anxiety could have increased the counter-regulatory hormones and possibly diminished the expected decrease of glucose levels. Additional large-scale studies are needed regarding specific subgroups of patients treated with HBOT, including patients with IDDM, children, those with myocardial infarction, etc. Since hyperbaric medicine is still a developing field in terms of indications for treatment, and co-morbidities and poly-pharmacy are integral aspects of our daily practice, it is important to study the groups of patients most vulnerable

Figure 1

Blood glucose before and after hyperbaric oxygen treatment (HBOT) or normobaric air (RA); mean (SEM) shown in three groups of patients: diabetics (DM), neurological injury (neuro) and healthy volunteers (HV)



to the physiological and biological effects of HBOT.

Conclusions

BGL may decrease during HBOT and accordingly it should be monitored before entering the chamber. However, this decrease in BGL should not necessarily be attributed to the hyperbaric environment per se.

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Hyperbaric oxygen therapy treatment for the recovery of muscle injury induced in rats

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Abstract

(Cervaens Costa Maia M, Camacho OF, Pinto Marques AF, Barata de Silva Coelho PM. Hyperbaric oxygen therapy treatment for the recovery of muscle injury induced in rats. *Diving and Hyperbaric Medicine*. 2013 December;43(4):222-225.)

Introduction: We evaluated the effect of hyperbaric oxygen treatment (HBOT) in the recovery of muscle injury in rats.

Materials and methods: Twelve female Wistar rats, weighing 200–250 g, were submitted to contusion of the right gastrocnemius muscle. Animals were then randomly assigned to an untreated control group or an HBOT-treated group. The HBOT group was given three, 60-minute sessions of HBOT at 253 kPa pressure at 24, 48 and 72 hours post injury. After the last session all animals were sacrificed and both gastrocnemius muscles removed, the left muscle as an internal control. Blood samples were taken for creatine phosphokinase (CPK). Using a standard traction technique, the muscles were analysed for their biomechanical properties: hardness, maximum elongation and maximum weight.

Results: Significant differences were found between uninjured and injured muscles and between untreated and HBOT groups in maximum weight and hardness: maximum weight in the non-treated group 18.27 ± 2.99 N versus 26.18 ± 2.84 N in the HBOT group ($P = 0.007$); hardness in the non-treated group $2.24 \pm 0.38 \times 10^3$ N m⁻¹ versus $3.19 \pm 0.32 \times 10^3$ N m⁻¹ in the HBOT group ($P = 0.001$). The difference in maximum elongation was not significant ($P = 0.793$). CPK was significantly different between the two groups (non-treated $6,445 \pm 387$ i.u. L⁻¹; HBOT $4,551 \pm 80$ i.u. L⁻¹; $P = 0.009$).

Conclusions: HBOT seems to play a positive role in the recovery of induced muscle injury in rats. However relevant, these results cannot be extrapolated to humans, for whom further clinical studies are warranted.

Key words

Hyperbaric oxygen, hyperbaric oxygen therapy, musculo-skeletal, injuries, research

Introduction

Muscle injury presents a challenging problem in traumatology and is very common in sports medicine.¹ Such injury may be a consequence of direct mechanical deformation (e.g., contusions, lacerations and strain) or of indirect causes (e.g., ischaemia and neurological damage).² More than 90% of muscle injuries are caused either by excessive strain or by contusion of the muscle.¹ A muscle suffers a contusion when it is submitted to a sudden, heavy compressive force, such as a direct blow.³ This can be classified as mild, a tear in a few muscle fibres with few symptoms that do not interfere with mobility; moderate, involving greater damage to the muscle affecting its function, or severe, a tear that extends across the entire cross-section of the muscle, preventing normal function.³

There is an emerging need for improved therapies that allow the injured athlete to return to competition faster and with a low risk of re-injury. The role of HBOT in the recovery of muscle injuries has been debated for several years, but remains poorly understood.⁴ The mode of action of HBOT is complex, the result of a number of physiological and pharmacological mechanisms based on elevation of both the partial pressure of oxygen and of the hydrostatic pressure.⁵ Considering the lack of consistent research on HBOT in soft-tissue injury, we aimed to evaluate the effect of HBOT in the recovery of muscle contusion inflicted to rats by measuring the biomechanical properties and haematological markers of muscle injury.

Materials and methods

ANIMALS

Twelve female rats, *Rattus norvegicus* albinos, Wistar-type, weighing 200–250 g, were studied. The animals were kept in the facilities of the Laboratory of the Faculty of Pharmacy, University of Porto, in collective cages with two animals per cage, at room temperature, receiving water and standard food ad libitum. All procedures were performed according to the FELASA recommendations for animal welfare and according to Portuguese legislation.

PROCEDURE FOR INDUCTION OF THE INJURY

Prior to the induction of the injury, the animals were anesthetized using 60 mg kg⁻¹ ketamine and 8 mg kg⁻¹ xylazine intraperitoneally. They were then positioned at the base of the lesion production equipment, in the ventral decubitus position, with their knee at maximal extension and ankle in neutral position (90°). In order to cause the lesion, a 171 g weight was released from a height of 102 cm onto the belly of the right gastrocnemius muscle in accordance with previous studies.⁶

Animals were then randomly assigned to two groups using an online programme, (www.randomizer.org). The control group received no treatment and the treatment group received three, 60-minute sessions of HBOT at 253 kPa pressure at 24, 48 and 72 hours after the injury. After the third session, all the

animals were sacrificed and the injured right gastrocnemius and the uninjured left gastrocnemius, which was used as an internal control, were surgically removed.

Blood samples were taken for analysis of creatine phosphokinase (CPK), a marker of muscle injury.⁷ Serum CPK was analyzed using a commercial enzymatic kit (Siemens, Flex Reagent Cartridge).

MECHANICAL TRACTION ASSAY

The removed muscle was biomechanically analysed using a standard traction machine (EMIC, DL 10000) to measure maximum elongation (ME), maximum weight (MW) and hardness (H). For the experiment, the machine was loaded with a charge of 50 kg force, and a pre-charge of 200 g was applied during 30 seconds for system accommodation. Afterwards, the assays were performed at a speed of 10 mm min⁻¹. ME corresponds to the maximum length of the muscle string before rupture; MW represents the maximum of imposed load before muscle rupture and H is a property obtained by the software (Tesc) that is determined by the slope of the line obtained in the elastic phase of the process.

STATISTICAL ANALYSIS

Statistical analysis was performed through the BioEstat@ program v. 2.0. Normality was checked with the Kolmogorov-Smirnov test. ANOVA analysis combined with a Bonferroni post-hoc test was used to evaluate differences between the groups. A pre-established significance level of $P = 0.05$ was used.

Results

For hardness, the internal control muscles had 3.92 ± 0.41 10^3 N m⁻¹, the non-treated group 2.24 ± 0.38 10^3 N m⁻¹ and the HBOT group 3.19 ± 0.32 10^3 N m⁻¹ ($P = 0.001$, Figure 1).

For maximum elongation, the internal control muscles had 13.40 ± 1.61 10^{-3} m, the non-treated group 10.91 ± 2.20 10^{-3} m and the HBOT group 11.70 ± 2.32 10^{-3} m ($P = 0.793$, Figure 2).

For maximum weight, the results were 32.23 ± 3.12 N for the internal control muscles, 18.27 ± 2.99 N for the non-treated group and 26.18 ± 2.84 N for the HBOT group (Figure 3, $P = 0.007$, Figure 3).

For CPK, a significant difference ($P = 0.009$) was found between the non-treated ($6,445 \pm 387$ IU L⁻¹) and the HBOT groups ($4,551 \pm 80$ IU L⁻¹).

Discussion

Not surprisingly, the non-injured muscle always had better results than the injured muscle, independently of whether or

Figure 1
Mechanical properties of muscle for hardness (10^3 N m⁻¹) in a non-injured control group and injured, non-treated and HBOT-treated groups; * $P = 0.001$ HBOT vs. non-treated

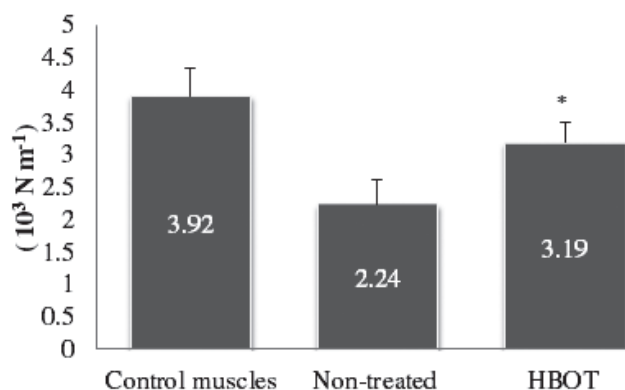


Figure 2
Mechanical properties of muscle for maximum elongation (10^{-3} m) in a non-injured control group and injured, non-treated and HBOT-treated groups; $P = 0.793$ HBOT vs. non-treated

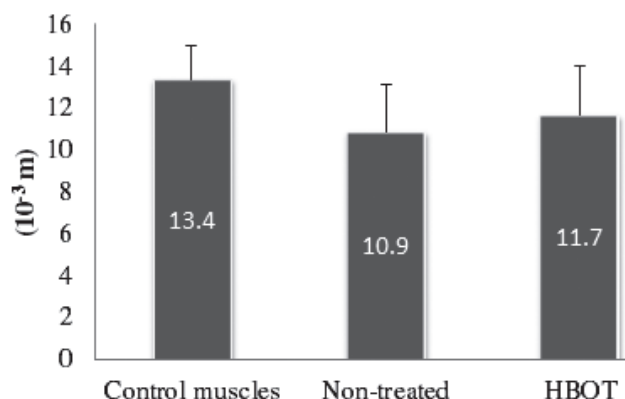
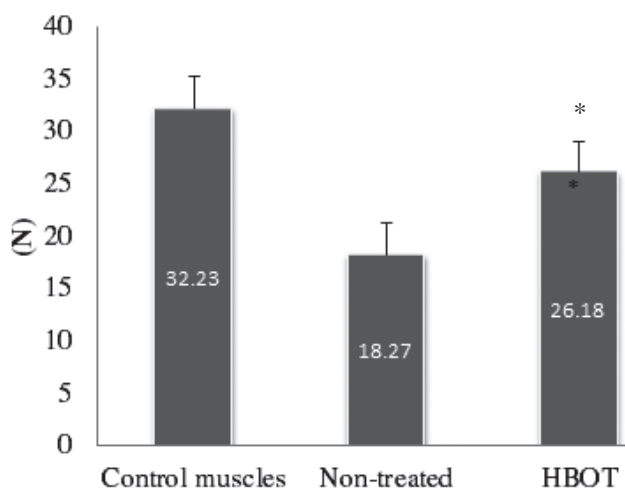


Figure 3
Mechanical properties of muscle for maximum weight (N) in a non-injured control group and injured, non-treated and HBOT-treated groups; * $P = 0.007$ HBOT vs. non-treated



not the animal received HBOT. This indicates that the injury protocol used was effective. The significant differences in hardness and maximum weight between the non-treated and HBOT groups, combined with the greater elevation of CPK in the untreated compared to the HBOT group indicates that HBOT had a positive effect on muscle injury recovery.

There is a lack of studies on the effect of HBOT on biomechanical properties of muscle injuries, particularly with regard to contusion. Therefore, we need to look at other types of muscle injury and the parameters measured in other studies. In a study of acute stretch injury of the tibialis anterior muscle in 18 rabbits, the animals were randomly assigned to two groups.⁸ One group received HBOT at 253 kPa for 60 minutes daily for five days starting 24 hours post injury, while the other had no treatment. Seven days after the injury, the deficit of ankle isometric torque in the HBOT group was less than that of the non-treated group and recovery was more complete, suggesting that HBOT may play a role in accelerating recovery after acute muscle stretch injury. Similarly in Sprague-Dawley rats, after 4 hours of ischaemia, the changes in levels of intracellular muscle compounds adenosine triphosphate, phosphocreatine and lactate were less in HBOT-treated rats than in untreated animals.⁹

In humans, a randomized double-blind trial studied 32 subjects with acute ankle sprains.¹⁰ Treatment consisted of three HBOT sessions at 203 kPa. HBOT did not influence ankle oedema, subjective pain indices, passive motion indices or the time to recovery. In a randomized, placebo-controlled trial in 66 patients with muscle soreness of the quadriceps, a control group, and sham, immediate and delayed HBOT groups were studied.¹¹ Delayed treatment and delayed sham were done at three or five days following injury using 203 kPa. Immediate HBOT patients had a better recovery than those treated at three days, while a delay of five days to HBOT provided the best results. In all groups, pain was similar.

If HBOT is applied within eight hours after tissue injury, it has been reported that accelerated recovery from soft-tissue injury results.¹² This was attributed to the known actions of HBOT, such as reduction of local hypoxia and inflammation, promotion of vasoconstriction, reduction of neutrophil adhesion, extinction of free radicals, control of oedema, increased leukocyte activity and promotion of procedures for the synthesis of collagen and blood vessel growth.⁵ In a Cochrane review examining the effect of HBOT on delayed-onset muscle soreness in untrained individuals, no conclusive evidence of benefit was found for HBOT on the speed of recovery in muscular pain.^{6,13-17} This review stated that more and larger randomized trials were needed to determine the role of HBOT in the treatment of sport injuries. High inspired normobaric oxygen has been reported to have positive effects on lymphatic vessel metabolism and oedema reduction.¹⁸ This has an interesting, potential

clinical application to enhanced protein removal from the site of injury by the lymphatic system.

In the present study, we used rats not only as an established animal injury model but also because, according to some authors, they exhibit musculoskeletal structure similar to humans.¹⁹ However, these results cannot be transferred to humans, but should give guidance to further research.

Conclusions

Using three biomechanical properties of injured muscle and CPK as a systemic marker of muscle injury, HBOT seems to play a positive role in the recovery of induced muscle contusion injury in rats. However, there is much we still need to understand about the use of HBOT in sports injury treatments. Therefore, more studies are warranted.

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Short communication

Comparison of venous glucose to finger-prick glucose in patients with diabetes under hyperbaric hyperoxic conditions: a pilot study

David McIlroy and Neil Banham

Abstract

(McIlroy D, Banham NDG. Comparison of venous glucose to finger-prick glucose in a diabetic population under hyperbaric hyperoxic conditions: a pilot study. *Diving and Hyperbaric Medicine*. 2013 December;43(4):226-228.)

Introduction: Blood glucose is commonly measured in diabetic patients undergoing hyperbaric oxygen treatment (HBOT) from a 'finger-prick' capillary sample. Although this method is an accurate reflection of venous glucose under normal conditions it has not been validated under hyperbaric, hyperoxic conditions.

Methods: Four patients with diabetes mellitus undergoing HBOT had venous blood samples drawn simultaneously with routine capillary samples before, during and immediately after three of four HBOT sessions. The Bland-Altman method of assessing agreement between these two measures was used separately for the three time periods.

Results: The relationship between venous and finger-prick glucose at room air was altered significantly by HBOT. The bias (finger-prick minus venous measurements) was significantly less than zero during the HBOT session but not immediately after completion of the session. Owing to the small sample size, the limits of agreement straddled zero at all time points, although the lower limit was close to zero during treatment (finger measurement appeared to be higher than venous measurement on room air and lower than venous undergoing HBOT).

Conclusion: Finger-prick capillary sampling may not be an accurate reflection of venous glucose during HBOT.

Key words

Diabetes, hyperbaric oxygen, blood sugar level, patient monitoring

Introduction

It has long been accepted as fact in hyperbaric medicine that blood glucose decreases in patients undergoing hyperbaric oxygen treatment (HBOT).¹⁻³ Several theories have been suggested, but none has gained widespread acceptance.

Our review of the relevant literature determined that many of the reports supporting a drop in blood glucose were only analysed by finger-prick sampling, which measures mixed subcutaneous capillary glucose.^{2,3} Further, the accuracy of glucometer assays have previously been validated under HBOT.^{4,5} Under normal conditions, these agree well with laboratory measured venous glucose.⁶

We hypothesised that subcutaneous capillary glucose may not be a reliable indicator of venous glucose under HBOT because blood flow in the subcutaneous tissues is reduced via vasoconstriction. This, combined with the hyperoxic state of the blood, will allow oxidative phosphorylation of glucose to continue for longer, and to a lower end point, and this process may be exaggerated in patients with already impaired micro-vascular blood flow.

Aims

Our aim was to determine the relationship between finger-prick capillary and venous glucose measured by glucometer with the patient breathing normobaric air and during HBOT.

Methods

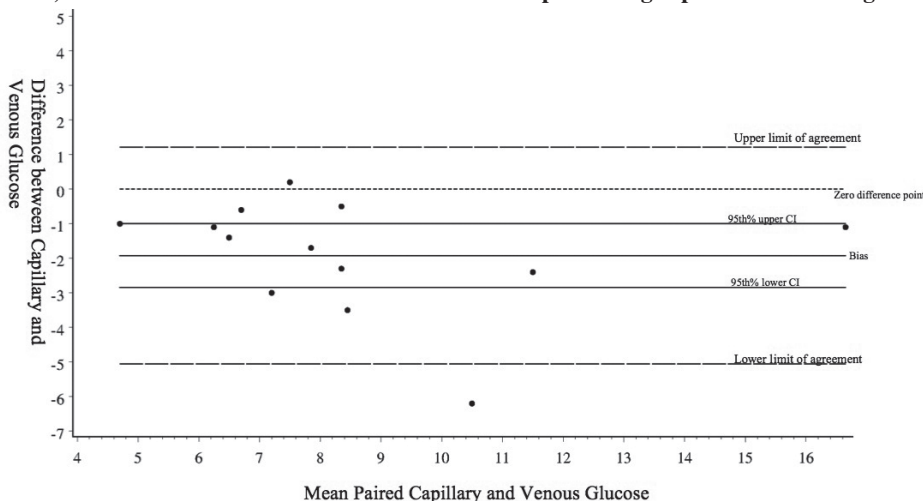
This study was approved by the South Metropolitan Area Health Service Human Research Ethics Committee, and performed in the Fremantle Hospital Hyperbaric Medicine Unit in June and July 2012. Adult patients over the age of 18 years with a diagnosis of diabetes mellitus for more than five years and receiving either insulin or oral hypoglycaemic agents were invited to participate in the study.

Prior to the first hyperbaric treatment session in the series an 18 gauge cannula was placed in an ante-cubital fossa vein. Then, in keeping with our standard practice, finger-prick glucose measurement samples were taken immediately prior to, during and immediately after the treatment session. The samples taken during treatment were obtained after 40 minutes of oxygen, just prior to the first air break. All treatments were at 243 kPa pressure. At the same time as the finger-prick sample, a venous sample was drawn. In collecting the venous sample the first 3 ml of blood was discarded, then 1 ml taken for glucose assay, then the cannula flushed with saline. The cannula remained in place for three days (in accordance with the Western Australia "Hospital in the home" service, <http://www.health.wa.gov.au/healthyathome/hith/index.cfm>), and samples were collected over three consecutive days where possible.

The samples were all analysed on the same Medisense Optium® glucometer, using Abbott® glucose test strips.

Figure 1

Comparison of measurements taken during the HBOT session. The dots show the actual data points, the three solid lines are the bias (central line), and its upper and lower 95% confidence limits, the two dashed lines are the upper and lower limits of agreement, and the dotted line marks the zero-difference point. Axis labelled “Difference” refers to finger-prick minus venous glucose values; axis labelled “Mean” refers to the mean of the paired finger-prick and venous glucose values.



This glucometer uses the glucose dehydrogenase reaction as the basis of its assay, which has previously been suggested to be most accurate under hyperbaric conditions.⁵ The decision not to send a venous sample to the laboratory was to eliminate any bias introduced by using a different assay.

Data analysis

The Bland-Altman method was used to assess any consistent bias between the two measures.⁷ Bland-Altman plots show the calculated bias, its upper and lower 95% confidence limits, and the upper and lower limits of agreement (LOA) between the two methods displayed graphically. The analysis was applied separately to the pre-, during- and post-treatment data. Statistical analyses were carried out with the SAS version 9.2 software (SAS Institute, Cary, NC, USA; 2008).

Results

Four patients were studied. Two subjects were sampled on three separate days and the other two over four consecutive treatment days. For both the pre- and post-HBOT figures, the biases on the Bland-Altman plots are close to zero, indicating

very close agreement. The venous measures were slightly lower than mixed capillary (i.e., the value for the difference is slightly positive), and the LOA straddle zero. During HBOT, the bias is negative and statistically significantly different from zero ($P = 0.0006$), indicating that the finger estimate of glucose is significantly lower than the venous measurement under hyperbaric conditions, and the LOA are wider apart (Figure 1). The LOA still straddle zero, but this is largely because of the small sample size, and the large variability in measurements. Table 1 shows the summary of the bias at each time point, its 95% confidence interval and the limits of agreement.

Discussion

Oxidative phosphorylation of glucose occurs only in the capillaries, so from first principles, arterial glucose should be higher than venous and mixed capillary should be between the two as it samples blood from the arteriolar and venular sides of the capillaries. This is supported by experimental work, with a difference in the region of 0.5–1.0 mmol L⁻¹ between venous and mixed capillary measurements.⁶ It has been assumed implicitly, although never validated, that this

Table 1

Characteristics of the difference between venous and finger-prick glucose; the bias is calculated as the mean of the difference (finger-prick minus venous), along with its standard deviation and 95% confidence interval. The *P*-value tests whether the bias is significantly different from zero; note that bias is significantly less than zero during treatment, while it is marginally positive pre-treatment and not significantly different from zero post-treatment.

Timing	<i>n</i>	Bias: mean (SD)	95% confidence interval for bias	<i>P</i> -value	Limits of agreement (Bland-Altman)
Pre-HBOT	13	0.56 (0.90)	0.02 to 1.11	0.0445	-1.21 to 2.33
During HBOT	14	-1.92 (1.60)	-2.85 to -1.00	0.0006	-5.06 to 1.21
Post HBOT	14	0.26 (0.75)	-0.18 to 0.69	0.2208	-1.21 to 1.72

agreement holds under hyperbaric oxygenation conditions, as the finger-prick method for blood glucose measurement has been accepted as an accurate surrogate measure of venous glucose.

These results suggest finger-prick mixed capillary blood glucose measurement may not be an accurate reflection of venous glucose measured during HBOT. During treatment sessions, the values obtained for finger-prick glucose samples read lower than for venous samples. It also appears the difference between the two values is less predictable under conditions of hyperbaric oxygenation than at room air and pressure (the larger standard deviation leads to LOA that are wider apart).

Although the cohort of patients was small, the line of best fit from the Bland-Altman plot prior to and post the HBOT session shows mixed capillary to be around 0.5 mmol L⁻¹ higher than venous, which is consistent with other larger studies. It then shows a very different relationship under HBOT where mixed capillary glucose appears to be significantly lower than venous glucose.

No symptomatic hypoglycaemic episodes were observed during the course of the study. In clinical practice, a low reading is accepted as representing true hypoglycaemia leading to the administration of glucose or glucagon. If, however, these measurements are not in close agreement, we may be raising the blood glucose, or tolerating high blood glucose levels unnecessarily, which may have a negative effect on wound healing. If the common in-chamber practice of assessing blood glucose with the finger-prick method is proven to exhibit bias, then a correction may need to be applied to the measurement (if possible), or some alternative method of blood glucose measurement may need to be used.

We suggest that any further work into the effects of HBOT on blood glucose will require measurement of central or venous glucose in addition to capillary sampling. A more definitive study is already planned to include non-diabetic volunteers to ascertain whether they behave in a similar fashion to diabetic patients. We would suggest that in-chamber finger-prick capillary blood glucose measurement is used with caution, as it does not appear to be an accurate or validated tool for blood glucose measurement during HBOT.

Conclusion

The relationship between subcutaneous mixed capillary glucose ('finger-prick') and venous glucose may be altered during HBOT. This alteration may be to such an extent that finger-prick glucose is not an accurate representation of venous glucose under hyperbaric conditions.

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Case reports

Acute coronary syndrome and decompression illness: a challenge for the diving physician

Marco Brauzzi, Fabio Andreozzi, Laura De Fina, Paolo Tanasi and Stefano Falini

Abstract

(Brauzzi M, Andreozzi F, De Fina L, Tanasi P, Falini S. Acute coronary syndrome and decompression illness: a challenge for the diving physician. *Diving and Hyperbaric Medicine*. 2013 December;43(4):229-231.)

Decompression illness (DCI) is a syndrome with diverse clinical manifestations but in which cardiac symptoms are rare. In the presence of cardiac symptoms, the necessity to rule out an acute coronary syndrome (ACS) which requires prompt treatment may result in delay to appropriate recompression treatment. We describe three cases with cardiologic symptoms referred to our centre by the Emergency Department (ED) of our facility. The first was a 48-year-old woman who lost consciousness during a dive and required cardiopulmonary resuscitation. The final diagnosis was acute myocardial infarction and the patient did not undergo recompression treatment. The second case was that of a 27-year-old man who complained of tachycardia, dyspnoea and vertigo soon after a dive. He was referred by helicopter ambulance and in the ED was diagnosed with new-onset atrial fibrillation. Recompression resulted in disappearance of his vertigo, and sinus rhythm was restored pharmacologically. The third case was a 43-year-old man, with a history of coronary artery disease, who had undergone coronary artery bypass grafting three years previously. After a repetitive dive without adequate decompression, he complained of crushing retrosternal pain and numbness in the upper left arm. All cardiovascular examinations were negative and the patient was recompressed, with resolution of his symptoms. Features to consider in arriving at the correct differential diagnosis in divers presenting with cardiac symptoms are discussed in the light of these three illustrative cases.

Key words

Decompression illness, decompression sickness, cardiovascular, diving accidents, case reports

Introduction

Decompression illness (DCI) is caused by the formation of air bubbles in blood or in tissues, during or after a reduction in environmental pressure (decompression). It includes two pathophysiological syndromes: arterial gas embolism (AGE) and the more common decompression sickness (DCS).¹ Recompression should occur as soon as possible to avoid deterioration and late recurrence.² In case of cardiac symptoms, distinguishing DCI from an acute coronary syndrome (ACS) is a potential challenge for the diving physician, given that both conditions require urgent but different treatments neither of which should be delayed.^{3,4} We describe three patients referred to our centre by the emergency department (ED) of our hospital. In all cases the dominant symptoms were cardiac.

Case reports

CASE 1

A 48-year-old female, with no risk factors for coronary artery disease (CAD), had passed a diving medical examination three months earlier. Weather conditions on the day of the incident were described as “nice”. At the beginning of the bottom phase of a scuba dive to 25 metres’ of sea water (msw), she lost consciousness without any prodromal symptoms.

Her dive buddy conducted an emergency controlled ascent to bring her to the surface. The helicopter ambulance arrived on the scene within 20 minutes. Oro-tracheal intubation was performed during medical evacuation to hospital. In the ED, blood analysis showed raised cardiac enzymes and a heart ultrasonography was performed. She suffered a cardiac arrest and advanced life support was initiated. Initially the team was unaware that the patient had lost consciousness early in the bottom phase of the dive, until a few minutes later the victim’s buddy described by phone the history of the accident. She was successfully resuscitated and admitted to the intensive care unit (ICU) on mechanical ventilation.

In the ICU, she had a large P_aO_2/F_iO_2 difference, with mild metabolic acidosis. She was haemodynamically unstable, requiring dopamine $10 \mu\text{g kg}^{-1} \text{min}^{-1}$; iv furosemide was administered. The first chest X-ray in the ICU showed pulmonary oedema with widespread alveolar involvement. Transthoracic echocardiography showed “left ventricle globally hypokinetic with normal size and a small pericardial effusion”. She was commenced on a heparin infusion. On the second day, a transoesophageal echo (TEE) showed an enlarged, akinetic left ventricular apex, aneurysm of the interatrial septum with a patent foramen ovale (PFO) and an ejection fraction of about 30%. Over the following days the patient rapidly improved, and two days after weaning from mechanical ventilation she was discharged from the

ICU. She was fully recovered in about 10 days. The final diagnosis was “*acute anterior myocardial infarction with acute pulmonary oedema in a patient with aneurysm of the interatrial septum and hypertrophic cardiac disease*”.

CASE 2

This diver was a 27-year-old male, referred by heliambulance for tachyarrhythmia and onset of intense vertigo about 30 minutes after a scuba dive to 56 msw for 18 min. Initially the patient complained of tachycardia and dyspnoea followed by the vertigo and nausea and vomiting. Personal history revealed thyroid disease and gastro-oesophageal reflux. The dive was performed within the limits of his diving computer profile. On arrival in the ED, new-onset atrial fibrillation with an average ventricular rate of 95 beats per minute (bpm) was diagnosed. Echocardiography revealed an aneurysm of the atrial septum. The patient was treated with a US Navy Treatment Table 6. During the treatment all neurological symptoms resolved. In the cardiology ward, after propafenone failed to convert his rhythm, amiodarone and heparin were administered, with reversal to sinus rhythm at a rate of 72 bpm. Follow-up TEE showed a type I Hanley interatrial aneurysm, with almost fixed prolapsing in the right atrium and a PFO with right-to-left shunting during a Valsalva manoeuvre. He received two further US Navy Treatment Table 5 (USN TT5) and five HBOT at 253 kPa for 90 min over the next seven days. Brain MRI (magnetic resonance imaging) was normal and he was asymptomatic when discharged.

CASE 3

A 43-year-old male presented himself to the ER in the evening after two dives, the first in the morning to 49 msw for 41 min with decompression, and the second in the afternoon also to 49 msw for 31 min with omitted decompression. He presented because of the onset of crushing retrosternal chest pain, tachyarrhythmia and left upper-arm paraesthesia, which recovered after a period of normobaric oxygen breathing. The patient's history revealed an episode of unstable angina, treated three years earlier with coronary artery bypass grafting (CABG), with subsequent complete recovery and good cardiac performance. Blood analysis and echocardiography showed no evidence of ACS. However, gas bubbles were seen in the right heart chambers on the echo. The patient was treated with a USN TT5 and discharged the following day after complete regression of his symptoms. He was advised to consult his cardiologist before resuming diving.

Discussion

CASE 1

Many cases of sudden death from cardiovascular disease with no prior history have been reported whilst diving,

cardiovascular events causing 20–30% of all deaths that occur while scuba diving.⁵ For many people, the real problem is that the first sign of CAD is a heart attack. In this case, the diagnosis was difficult because of the clinical condition of the patient and the initial lack of information on the dynamics of the accident. Therefore, the most probable diagnosis in the early phase was DCI. Luckily the phone call received from the diving centre explained the situation and described the onset of symptoms. Otherwise we were ready to treat her in the hyperbaric chamber.

CASE 2

Atrial fibrillation is the most common cardiac dysrhythmia. It may be asymptomatic, but is often associated with palpitations, fainting, chest pain or congestive heart failure. However, in some people, atrial fibrillation is caused by either idiopathic or benign conditions. Paradoxical embolization occurs when gas that has entered the venous circulation migrates to the systemic arterial circulation, leading to signs and symptoms of AGE.⁶ The two mechanisms by which this can occur are migration of gas through a right-to-left shunt, as in patents with PFO, or overwhelming the pulmonary capillary filtration system. These bubbles may embolize to the whole body, including the coronary circulation. The clinical manifestations are dependent on the volume of gas that enters the vasculature and the rate of entrainment of gas. Generally from a cardiological viewpoint, symptoms of right heart strain may develop or, as cardiac output declines, tachyarrhythmia and hypotension may develop, as happened in this case. Cardiac symptoms of DCS do not receive enough consideration. It is suggested that, whenever possible, an electrocardiogram should be performed in the basic evaluation of suspected DCS.

CASE 3

Severe DCI is characterised by central nervous system and pulmonary symptoms and circulatory problems such as hypovolaemic shock.¹ Pain is reported in only about 30% of cases. Because of the anatomical complexity of the central and peripheral nervous systems, signs and symptoms are variable and diverse. Symptom onset is usually rapid, especially in severe cases of DCI, but may be delayed for up to 36 hours after the dive.

In the 2005 Diver Alert Network Report, more than 14% of the fatalities reported had a chronic history of high blood pressure and/or heart disease.⁷ Obesity, another factor reported in 55% of fatalities, is connected to heart disease and hypertension, with resulting links to poor health and poor exercise tolerance. In combination with other contributing factors, cardiopathy can increase the risk of a severe or fatal dive incident.

An individual who has undergone CABG or angioplasty may have suffered significant cardiac damage before the surgery.

The post-operative cardiac function of individuals dictates their fitness for diving. In patients who have had open-chest surgery, after a period of stabilization and healing (6–12 months is usually recommended), a thorough cardiovascular evaluation is needed prior to being cleared to dive. Patients should be free of chest pain and have normal exercise tolerance, as evidenced by a normal stress test (13 mets or stage 4 of the Bruce protocol). If there is any doubt about the success of the procedure or about the condition of the coronary arteries, the individual should refrain from diving.

In Case 3 the problem was differential diagnosis between myocardial infarction and DCI. The blood analysis and cardiac evaluation, which proved negative, resulted in a delay of about three hours in starting recompression treatment. This diver resumed diving activity after his CABG surgery without prior clearance from a diving physician.

The onset of cardiac symptoms after a dive, particularly in patients with a clinical history of CAD does not automatically rule out the diagnosis of DCI, just as neurological symptoms do not rule out an ACS. Therefore, the assessing physician needs to take into consideration:^{3–7}

- timing of the onset of symptoms after the dive;
- the realistic possibility of DCI (based on the time and depth of the dive, the necessity for decompression, how the decompression was performed);
- physical activity at the time of onset of symptoms;
- severity of symptomatology and its development (i.e., improving, stable or deteriorating);
- the diver's previous medical history;
- whether similar or the same symptoms have occurred in the past after dives.

In some cases, a differential diagnosis may be difficult, as in these cases. Whilst the predominant symptoms were cardiac, only in one case was there no need for recompression.

Conclusions

So we may conclude that:

- DCI may sometimes onset with cardiac symptoms;
- it is not true that all cardiac symptoms at the end of a dive are the result of DCI;
- a patient with a previous CABG must be carefully evaluated before starting recompression treatment;
- it is mandatory that a thorough cardiological assessment is performed on all patients presenting with symptoms of DCI, and maintained for at least 48 hours.

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Patient consent

All three patients, one being a foreign national, were lost to follow-up, and all attempts to contact them have failed.

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The diving doctor's diary

Editor's note:

Pulmonary barotrauma (PBT) and its consequences are well recognised in scuba diving, but less so in association with breath-hold diving. The same may be said for iatrogenic PBT and arterial gas embolism (AGE), and failure to recognise these may (and sadly often does) lead to tragic consequences. The five cases reported in the following articles, and a later letter to the Editor regarding retrograde venous gas embolism have been put together to remind clinicians to always be on the lookout for these complications. I have myself managed breath-hold divers who have taken a breath from a scuba regulator at depth, leading to AGE, and iatrogenic cases similar to the one reported by Janisch et al.

Pneumomediastinum or lung damage in breath-hold divers from different mechanisms: a report of three cases

Akin Savaş Toklu, Mustafa Erelel and Abdullah Arslan

Abstract

(Toklu AS, Erelel M, Arslan A. Pneumomediastinum or lung damage in breath-hold divers from different mechanisms: a report of three cases. *Diving and Hyperbaric Medicine*. 2013 December;43(4):232-235.)

Normally pulmonary over-inflation is not an issue during breath-hold diving, in contrast to lung squeeze. Compared with compressed air diving, pulmonary barotrauma is rare in breath-hold diving. Several mechanisms can lead to an increase in intrathoracic pressure in breath-hold diving that may cause alveolar rupture. Here we report three cases of pulmonary barotrauma in breath-hold diving. Using high-resolution chest tomography, bullous damage in Case 1, and pneumomediastinum in Cases 2 and 3 were detected. Transient neurological symptoms in Cases 1 and 2 suggested cerebral arterial gas embolism. The mechanisms that caused intrapulmonary overpressure were, respectively, lung packing ('buccal pumping'), considerable effort and straining at depth, and breathing compressed air at depth and ascending without exhaling. All three cases recovered without specific treatment such as recompression.

Key words

Breath-hold diving, freediving, pulmonary barotrauma, arterial gas embolism, cerebral arterial gas embolism (CAGE), buccal pumping, case reports

Introduction

Breath-hold diving is associated with a variety of physiological responses induced by immersion, apnea and lung compression.¹ Normally pulmonary over-inflation is not an issue during breath-hold diving, in contrast to lung squeeze.² Compared with compressed air diving, pulmonary barotrauma (PBT) is rare in breath-hold diving. Nevertheless, several mechanisms can lead to an increase in intrathoracic pressure.² Alveolar rupture can cause pneumothorax, pneumomediastinum, surgical emphysema and arterial gas embolism (AGE) and may occur when intrapulmonary pressure is higher than environmental pressure, such as in divers and aviators or airline passengers and mechanically ventilated patients.^{3,4} PBT may also be caused by repeated bearing down to increase abdominal pressure, sneezing, vomiting and oesophageal rupture. PBT is especially a risk for divers using scuba or surface-supply breathing apparatus (SSBA). Gas may escape from ruptured alveoli to the interstitial space, tracking along perivascular sheaths or entering the pulmonary circulation via torn pulmonary vessels to reach the left heart, leading to AGE.⁵

Cerebral (CAGE) or coronary AGE are the most dangerous complications of pulmonary barotrauma and CAGE is a major cause of mortality in diving.⁶

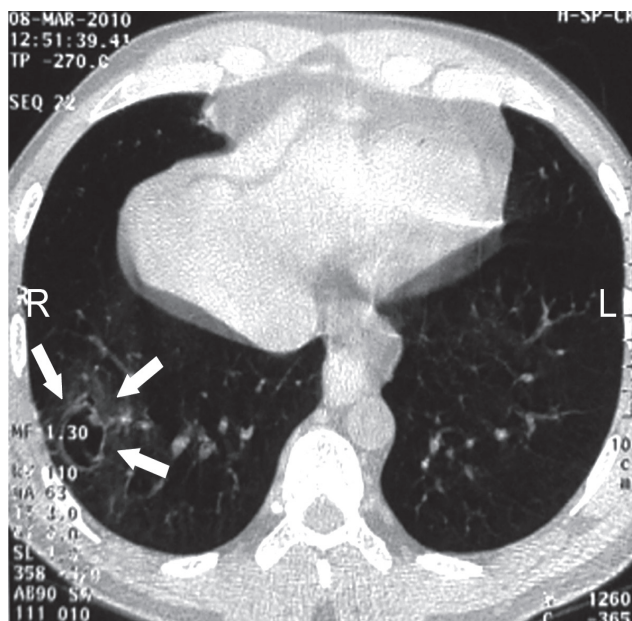
We report three cases of breath-hold (BH) divers who suffered PBT from three different but well-documented mechanisms and presented between 2010 and 2012. Patients gave their verbal consent for their cases to be reported.

Case reports

CASE 1

A 40-year-old, otherwise healthy, male, competitive breath-hold diver, who was in the habit of lung packing (glossopharyngeal insufflation or 'buccal pumping') prior to breath-holding, made a dive in a swimming pool to a depth of approximately 1.5–2 metres of water for dynamic apnea training. He performed lung packing for 20 seconds prior to the dive. He felt dizziness, nausea and numbness all over his body 10 seconds after his dive started, and was unable to control the right side of his body and had visual disturbances

Figure 1
Case 1: a single HRCT cut showing bullous damage
in the right lung



when he surfaced. After about an hour, all symptoms had resolved spontaneously. On presentation two days after the incident, physical examination was unremarkable except for rales at the right lung base. High-resolution chest tomography (HRCT) revealed bullous damage in the right lower lobe (Figure 1). Cerebral magnetic resonance imaging (MRI) was normal. He was advised not to dive until a repeat HRCT three months after the incident, which revealed no pathology. He was advised not to use manoeuvres such as lung packing that increase transpulmonary pressure.

CASE 2

An 18-year-old, male breath-hold diver made four dives to about 18 metres' sea water (msw) depth, each lasting 45–60 seconds, with 1–2 minute surface intervals. Then after about an hour, he made another ten dives to 6–7 msw depths to release a rope that was stuck underneath a rock at the bottom. This involved considerable effort and straining. He did not perform lung packing before any of the dives. He had pain and a sensation of pressure in his chest for 30 minutes after his last dive and noticed swelling around his neck that was crepitant on palpation. He was diagnosed as having a pneumomediastinum, confirmed by HRCT (Figure 2). The symptoms disappeared within two days without treatment. Repeat HRCT 20 days after the incident revealed no pathology.

CASE 3

An otherwise healthy, 36-year-old man made three breath-hold dives to 3–5 msw without any problems. He made a final dive to 9 msw, during which he breathed

Figure 2
Case 2: a single HRCT cut showing mediastinal
and cervical emphysema

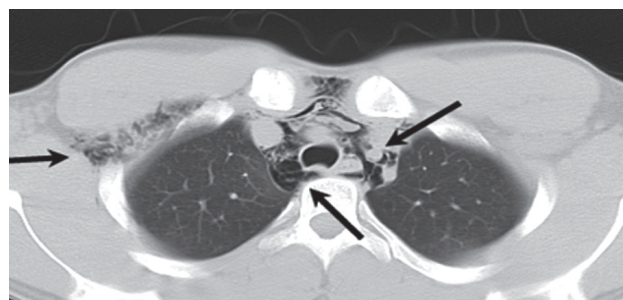
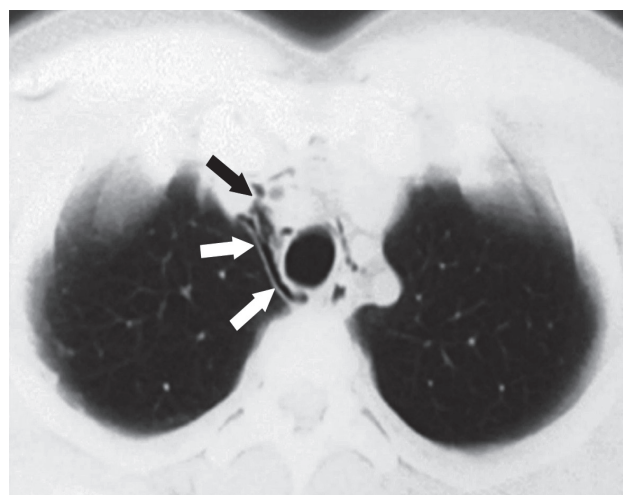


Figure 3
Case 3: a single HRCT cut showing mediastinal emphysema



from a friend's scuba 'octopus' regulator before ascending to the surface. During the ascent, he did not exhale and he felt pressure in his chest at 4–5 msw. Some air escaped from his mouth involuntarily when he was about to surface. He felt retrosternal pain and jerking and abnormal sensation over the right side of his torso and leg when he reached the surface. He was lifted into the boat with difficulty, where his right leg felt weak and he was dysarthric, which subsided within about 10 minutes. His symptoms, which clearly indicated CAGE, almost completely disappeared within 30 minutes except for the chest pain. Thirty minutes after the incident, oxygen via nasal prongs and an intravenous infusion of electrolytes were commenced and he was given dexamethasone 8 mg intramuscularly. The patient reported that his retrosternal pain decreased within 30 minutes following the initiation of this treatment.

A diving physician recommended by phone that he have HRCT and be transferred on 100% oxygen for recompression treatment on the presumptive diagnosis of PBT/CAGE. HRCT was normal. He was discharged the same day to present the next day to the

Department of Underwater and Hyperbaric Medicine, İstanbul Faculty of Medicine, since he still had slight chest pain while breathing. There were no pathological findings on physical examination. However, mediastinal emphysema was detected on his HRCT (Figure 3). Repeat HRCT three weeks later was normal. He was advised not to resume breath-hold diving for six months.

Discussion

The first diver was a sport breath-hold diver who competed in the discipline of dynamic apnea (underwater swim in a swimming pool with or without fins for as great a distance as possible on a single breath-hold). For this, breath-hold time is critical, and some athletes perform various manoeuvres, such as hyperventilation or lung packing, to extend their breath-hold time. Vital capacity can be increased by more than 30% by lung packing, which can increase transpulmonary pressures by up to 80 cm H₂O, and lung rupture may occur.⁷ There are several reports of lung damage after lung packing.⁷⁻⁹ In our first case, the initial symptoms were neurological and it is likely this was the result of CAGE from air entrainment into the pulmonary veins from an area of ruptured alveoli. CAGE may be fatal, so it makes sense to discourage BH divers from performing lung packing before diving. The bullous damage in the lung healed within three months with no bulla being detected on HRCT. There are a few case reports showing spontaneous collapse of bullae in non-divers in long-term follow up.^{10,11} On the other hand, the cystic lesions did not resolve in long-term follow up in divers with pulmonary barotrauma in another study in which the authors assumed that the lung cysts or blebs were pre-existing conditions which had led to the pulmonary barotrauma.¹² In another series of three cases, bullae were seen to increase in diameter, seemingly related to diving.¹³ In our case, the bullous lesion was likely owing to trauma from overpressure.

In the second case, the cause of the diver's lung injury was probably straining against a closed glottis during his efforts to free the jammed rope.¹⁴ Underwater tasks that require physical effort and straining should not be done by breath-hold diving but rather using scuba or SSBA.

Lung over-inflation is not normally expected in breath-hold diving, but the third diver practised something completely outside of BH diving rules in breathing from scuba equipment at depth. Having done so, if the diver continues to hold his breath, the decrease in ambient pressure during ascent results in an increase in intrathoracic gas volume and lung rupture, which may be fatal. This diver was not a scuba diver and, therefore, he was not aware of the risk of breathing compressed air at depth and then ascending whilst still holding his breath.

For the same reason, free emergency ascent was abandoned as part of scuba training about 20–25 years

ago.¹⁵ Free ascent from 18 msw is still part of submariner training in the controlled environment of a submarine escape training tank. The risks associated with this training are well documented.^{16,17}

In PBT cases, 100% oxygen administration is an important component of first aid. However, nasal prongs are an unsuitable mode of delivery as they provide considerably less than 100% oxygen. Oxygen should be given by mask with reservoir bag or other 100% oxygen delivery system in such cases. Many PBT cases recover spontaneously, as in all three of these divers.¹⁸ However, it is important that, where there is a suspicion of PBT, a recompression facility should be sought since the diver's clinical condition may deteriorate secondarily and sufficiently to be life-threatening. Consideration should be given to recompression treatment for all PBT cases with neurological symptoms or signs, however transient, as in Cases 1 and 3. At the very least, the diver should be monitored carefully.

Symptoms and signs of PBT usually appear within minutes to an hour after the dive.⁴ While symptoms like retrosternal pain and coughing may point to pneumomediastinum, and CAGE may cause neurological symptoms, sometimes PBT cases may be asymptomatic. HRCT is a valuable tool in determining the extent and presence of mediastinal emphysema, as plain chest radiographs may miss a small pneumomediastinum.¹⁹

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Spontaneous pneumomediastinum in an 11-year-old boy after a shallow breath-hold dive

Maija Laitila and Vesa Eskola

Abstract

(Laitila M, Eskola V. Spontaneous pneumomediastinum in an 11-year-old boy after a shallow breath-hold dive. *Diving and Hyperbaric Medicine*. 2013 December;43(4):235-236.)

Spontaneous pneumomediastinum is caused by pulmonary barotrauma due to transiently increased intra-alveolar and intra-bronchial pressure. The most frequent triggers of spontaneous pneumomediastinum in children are asthma and manoeuvres creating forced expiration. It has been rarely associated with breath-hold diving. Chest pain and dyspnoea are the main symptoms, and the diagnosis can be confirmed by chest X-ray. The treatment of choice is oxygen, analgesics and monitoring the patient. The recurrence rate is low. The main differential diagnoses of spontaneous pneumomediastinum are oesophageal perforation and pericarditis. We report a case of an 11-year-old boy with no substantial medical history, who tried to breath-hold in shallow water for as long as possible. After diving, he felt dyspnoea and chest pain. Chest X-ray revealed pneumomediastinum and subcutaneous emphysema. The patient was admitted to the PICU for observation and was discharged after two days' follow up. Spontaneous pneumomediastinum in children may be more common than thus far acknowledged. It requires a high index of suspicion and should be considered in all children with acute chest pain.

Key words

Breath-hold diving, pulmonary barotrauma, children, radiological imaging, case reports

Introduction

Spontaneous pneumomediastinum is caused by pulmonary barotrauma (PBT) due to transiently increased intra-alveolar and intrabronchial pressure. The most frequent triggers of spontaneous pneumomediastinum in children are asthma and manoeuvres creating forced expiration.^{1,2} It has been very rarely associated with breath-hold diving. We report the case

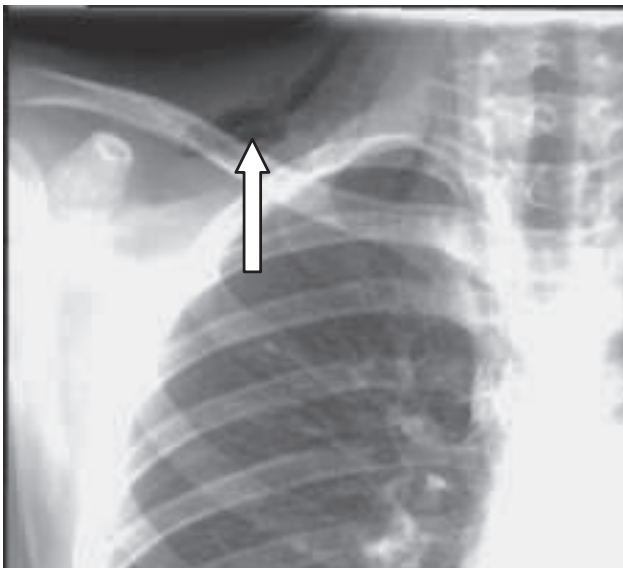
of a child with evidence of PBT after a breath-hold dive.

Case report

An 11-year-old boy, with no substantial medical history, tried to breath-hold in shallow water (0.5–1 metre depth) for as long as possible. Immediately after diving he felt dyspnoea and right-sided chest pain. The pain settled but

Figure 1

Chest radiograph with arrows demonstrating air outlining the left heart border and subcutaneous emphysema in the neck



did not disappear completely. The pulse was elevated and irregular, and there was a sense of constriction around the neck. Deep breathing increased the symptoms. On admission, his general condition was stable and all vital signs within normal limits. Physical examination was normal, except for pain on palpation in the right fourth intercostal space. ECG, blood count, C-reactive protein, troponin T and blood gas analysis were within normal limits. The patient was admitted to the PICU for observation. Chest X-ray showed pneumomediastinum and subcutaneous emphysema on the right side of the neck (Figure 1). Next day, he was asymptomatic and pneumomediastinum had resolved. He was discharged after two days, and chest X-ray 10 days later was normal. Parental permission was given to report his case.

Discussion

The detection of spontaneous pneumomediastinum requires a high index of suspicion and should be considered in all children with acute chest pain. Air leaking from an alveolar rupture may dissect along the tracheobronchial tree, leading to cervical and mediastinal emphysema. The most frequent triggers of spontaneous pneumomediastinum in children are asthma, vomiting, shouting, coughing, foreign body aspiration, breath-holding and intense sport activities, but it is rarely associated with free diving.^{1,4} The incidence of spontaneous pneumomediastinum has ranged from 1:800 to 1:42,000 children treated in hospital emergency units.^{1,2} The occurrence is bimodal with peak incidences in children younger than seven and older than 15 years.²

The combination of chest pain, dyspnoea and subcutaneous emphysema in the neck and/or supraclavicular fossae is found in 40% cases, though pain is present in 50–90 %.¹ Other possible symptoms are weakness, back and shoulder

pain, swollen neck, sore throat, dysphagia, torticollis and abdominal pain.^{1–3} The diagnosis can be confirmed by chest X-ray, which may show radiolucent streaks of air outlining various structures, tracking vertically, and subcutaneous emphysema.⁵ A double image along the left heart border, where the two layers of the pericardium are separated by air, is a classic and sometimes the only positive radiological sign of pneumomediastinum. Follow-up X-rays are indicated only if the patient's condition alters.²

The main differential diagnoses of spontaneous pneumomediastinum are oesophageal perforation and pericarditis. A concomitant pneumothorax, or underlying asthma, pulmonary embolism or pneumonia should be considered. Rare complications include pneumopericardium, tension pneumothorax and diffusion of air elsewhere.^{1,2}

The treatment of choice is oxygen, analgesics and monitoring of the patient, and the recurrence rate is low. It resolves within three days to two weeks. Patients should avoid diving and manoeuvres creating forced expiration to lessen the risk of barotrauma.^{1–5} The presence of asthma, wheeze or airway hyperresponsiveness should prevent children and adolescents from diving, but this boy with spontaneous pneumomediastinum with no substantial medical history has no absolute contra-indications to scuba diving in the future.⁶

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Cerebral air embolism after pleural streptokinase instillation

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Abstract

(Janisch T, Siekmann U, Kopp R. Cerebral air embolism after pleural streptokinase instillation. *Diving and Hyperbaric Medicine*. 2013 December;43(4):237-238.)

Iatrogenic pulmonary barotrauma and cerebral arterial gas embolism (CAGE) may complicate a variety of medical procedures, such as certain types of surgery, drug administration through thoracic drainage, pneumoperitoneum, cystoscopy, bronchoscopy, etc. Hyperbaric oxygen treatment following the guidelines for CAGE in diving is the treatment of choice. Pleural streptokinase instillation is a common treatment for parapneumonic pleural effusion and may lead to CAGE. We present such a complication in a 79-year-old woman with a left-sided empyema. Neurological recovery was reasonable, but a left hemiparesis persisted. Prompt treatment of CAGE is necessary to avoid permanent injury and severe disability.

Key words

Pulmonary barotrauma, cerebral arterial gas embolism (CAGE), hyperbaric oxygen therapy, case reports

Introduction

Iatrogenic pulmonary barotrauma and arterial gas embolism (AGE) may complicate a variety of medical procedures.¹⁻⁵ We present a case of cerebral AGE (CAGE) in a patient undergoing pleural streptokinase instillation.

Case report

A 79-year-old woman, with a medical history of arterial hypertension, hypothyroidism after radioiodine-therapy 30 years before, an untreated umbilical hernia, diverticulitis and fractures of the humerus and pelvis two months before, was admitted to hospital because of a decline in her general health. Chest X-ray showed left-sided pneumonia and empyema. Antibiotic therapy with tazobactam and piperacillin was started and the pleural effusion was relieved with a dual-sump drain. Next day, chest X-ray showed a persistent pleural effusion, so 250,000 i.u. streptokinase were instilled into the pleural space via the drain. Immediately after instillation, the patient showed a marked reduction of consciousness associated with a right lateral gaze.

She was intubated and mechanically ventilated. Cranial computer tomography (CT) showed multiple pockets of cerebral air (Figures 1 and 2), whilst a thoracic CT showed no pneumothorax (Figure 3). Because of the suspected diagnosis of CAGE with the need for hyperbaric oxygen treatment (HBOT), transfer to a hospital with a hyperbaric chamber was arranged. Organisation and transfer took about 24 hours. At the hyperbaric centre, the sedated, intubated and mechanically ventilated patient was treated with a US Navy Treatment Table 6 (USN TT6) with extensions (in total, 3 hours at 280 kPa and 4 hours at 190 kPa). After HBOT, repeat cranial CT showed no more cerebral air. Further HBOT was not undertaken because of the non-availability of hyperbaric staff. After reduction of sedation in the intensive care unit, the patient showed inadequate awaking, a left lateral gaze and focal seizures. An EEG showed status epilepticus,

which responded to phenytoin, topiramate and levetiracetam. The patient underwent tracheostomy because prolonged weaning from mechanical ventilation was anticipated. A further cranial CT showed ischaemic infarcts in the areas of the right anterior and middle cerebral arteries; chest CT showed no changes from the previous CT.

Over several weeks her neurological status improved and she was discharged to a neurological rehabilitation unit six weeks after the incident. At that time, she had a persistent left hemiparesis, predominantly of the arm, and could speak, sit and walk up to 150 metres with the assistance of a wheeled walker. She had no more seizures under anti-epileptic medication.

Discussion

AGE is a rare but well-recognised complication of certain types of surgery, intravascular radiological procedures, drug administration through thoracic drainage, pneumoperitoneum, cystoscopy, bronchoscopy, gunshot or penetrating injuries of the thorax, etc.¹⁻⁵ Clinical signs should lead to diagnosis. In some cases, the cranial CT shows no gas in the cerebral circulation as gas is usually pushed through the cerebral circulation. Sometimes persistent air bubbles may be too small to detect with routine CT.⁶

Whether the cause is iatrogenic or diving accidents, HBOT reduces bubble size and enhances elimination of any inert gas.³ Furthermore, the high oxygen partial pressure can maintain aerobic metabolism in poorly perfused tissue by diffusion and reduces cerebral oedema.⁷ Prompt HBOT could lead to a *restitutio ad integrum*.^{3-5,8} Similarly to the treatment of CAGE in divers, a second (or more) HBOT should follow the first within 24 hours if symptoms persist.⁹ In our case, an extended USN TT6 was delayed 24 h and further treatment was not possible for logistic reasons. The cerebral ischaemic infarcts reported on follow-up CT might have been avoided if treatment could have been commenced

Figure 1
CT of the brain, arrow shows air pocket

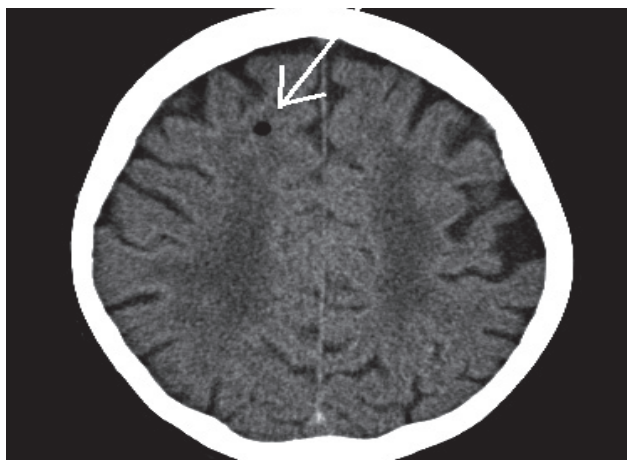
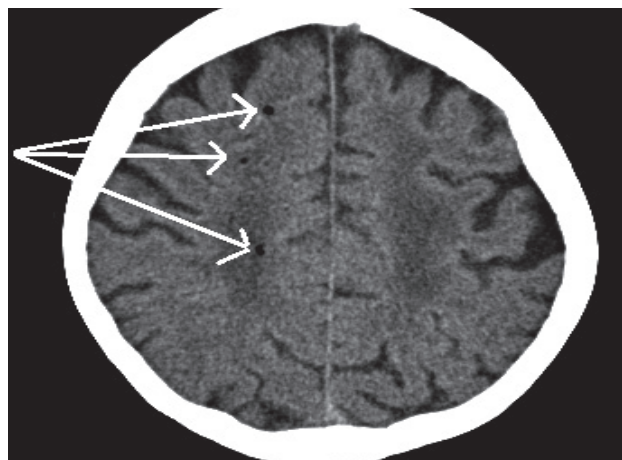


Figure 2
CT of the brain, arrows show air pockets



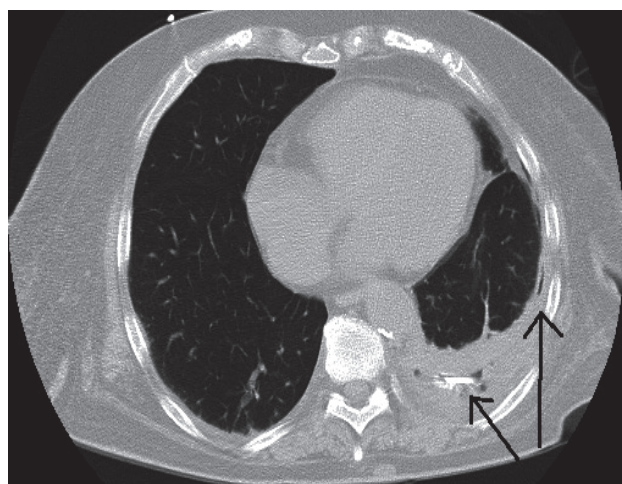
earlier. Unfortunately in Germany there is a lack of hyperbaric facilities with 24-hour emergency intensive care capability as a result of governmental policies. Nevertheless, and not withstanding that studies suggest that clinical outcome is better the shorter the delay to recompression following CAGE, HBOT is the only effective treatment for CAGE.⁹ The take-home message is that treatment of CAGE with HBOT has to happen as soon as possible, because “time is brain”.

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Figure 3

CT of the thorax, showing left-sided empyema, arrows show some air pockets and a chest drain



from: <http://www.gtuem.org/wcms/ftp//g/gtuem.org/uploads/leitlinietauchunfall2011awmftemplateleitlinienreport.pdf>

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The world as it is

Managing scientific diving operations in a remote location: the Canadian high Arctic

Martin DJ Sayer, Frithjof C Küpper, Pieter van West, Colin M Wilson, Hugh Brown and Elaine Azzopardi

Abstract

(Sayer MDJ, Küpper FC, van West P, Wilson CM, Brown H, Azzopardi E. Managing scientific diving operations in a remote location: the Canadian high Arctic. *Diving and Hyperbaric Medicine*. 2013 December;43(4):239-243.)

Global climate change is expected to alter the Arctic bioregion markedly in coming decades. As a result, monitoring of the expected and actual changes has assumed high scientific significance. Many marine science objectives are best supported with the use of scientific diving techniques. Some important keystone environments are located in extremely remote locations where land-based expeditions offer high flexibility and cost-effectiveness over ship-based operations. However, the extreme remoteness of some of these locations, coupled with complex and unreliable land, sea and air communications, means that there is rarely quick access (< 48 h) to any specialized diving medical intervention or recompression. In 2009, a land-based expedition to the north end of Baffin Island was undertaken with the specific aim of establishing an inventory of the diversity of seaweeds and their pathogens that was broadly representative of a high Arctic marine environment. This account highlights some of the logistical considerations taken on that expedition; specifically it outlines the non-recompression treatment pathway that would have been adopted in the event of a diver suffering decompression illness.

Key words

Scientific diving, remote locations, decompression illness, injuries, first aid, oxygen, medical kits

Introduction

The polar bioregions are the areas of the globe most sensitive to changes in the climate; major regional modifications are already being observed in polar marine ecosystems that have experienced warming.¹ A pole-ward migration of existing and invading species has the growing potential to alter community composition and species abundance. Warming of areas of the polar oceans has already had negative effects on community composition, biomass and distribution.¹ Environmental changes of substantial magnitude have the potential to cause significant ecosystem impact and there has been mounting pressure on the scientific community to understand the present conditions in order to monitor departures from the baseline and to predict any potential consequences. In the polar seas, shallow coastal areas are extremely vulnerable to the effects of climate change principally because of alterations in the types and behaviours of sea ice.² Scuba diving is a highly productive and cost-effective research tool in such environments.³

DIVING ACTIVITIES IN POLAR REGIONS

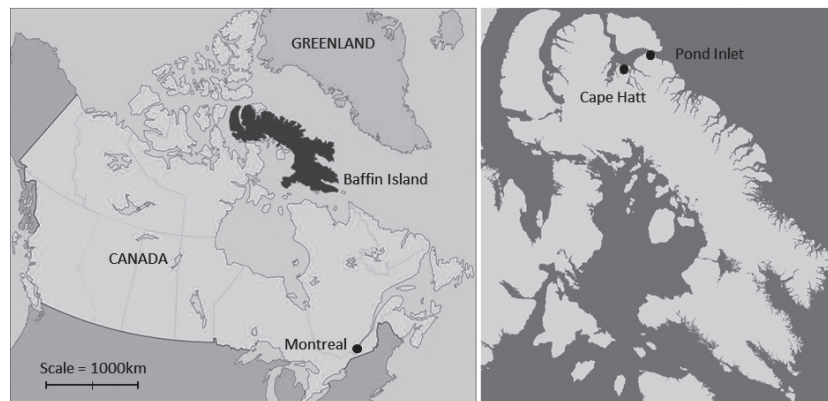
Although both polar regions are typified by having vast surface areas and coastlines but with minimal inhabitation and hugely limited communications, they differ markedly in how scientific field operations are supported. Nations actively engaged in scientific research in the Antarctic tend to maintain permanent or semi-permanent support infrastructure with dedicated methods of transportation.

Scientific diving operations in Antarctica are well-resourced and tend to occur close to the main bases which, in most cases, have doctors on-site with good medical facilities and recompression chambers.^{4,5} There are relatively few scientific bases in the high Arctic and this, in combination with the remoteness and limited communications of the region, means that most marine science expeditions to the area tend to be research vessel-based. There are few research vessels that are capable of working at high latitudes, they are expensive to maintain and they may be limited in their operational range. There are definite logistical benefits to conducting shallow coastal research in the high Arctic using land-based diving. Decompression illness (DCI) is always a potential end point to any diving activity, and there are additional challenges when diving in cold environments.⁶ The employment of diving in a remote location will either need to be supported by a portable recompression chamber or the risk management process will need to take into account alternative methods of treating DCI.

SENSITIVITY OF HIGH-LATITUDE COASTAL ECOSYSTEMS

Macroalgae are an essential element of coastal ecosystems and the degradation of macroalgal vegetation communities can affect the entire ecosystem in an area.⁷ Benthic marine algae make significant contributions to high-latitude coastal primary productivity and energy fluxes, exceeding or equalling the production of primary producers in more temperate systems.⁸ In the Arctic, floristic knowledge of

Figure 1
Map showing the location of Baffin Island (dark shading) in respect to the nearest recompression chamber at Montreal;
the inset of Baffin shows the locations of Pond Inlet and the Cape Hatt field camp



the diversity of macroalgae is fragmented, particularly in the American Arctic.⁹ Macroalgae of the Arctic region are considered to be very sensitive to a reduction in ice coverage because the life histories of many macroalgae are regulated by temperature and photo-regimes and the periodic physical presence and absence of sea ice, to a range of impact depths, may drive recruitment of some taxa.^{10–13} There are predictions that seaweed composition and abundance will change with elevated temperatures; however, those predictions vary regionally and without a detailed baseline inventory it will not be possible to measure or monitor change as or if it happens.^{7,11,13}

It is becoming increasingly accepted that pathogens impact the overall structure of natural communities as well as the flow of energy and matter within ecosystems.¹⁴ Previous studies have suggested that climate change may be contributing to increasing numbers of pathogenic epidemics that have the potential to exert devastating effects on ecosystems.¹⁵ However, many of these conclusions are inferred or cannot be explained with any certainty because baseline estimates for pathogen populations are lacking. In particular, there is an almost complete gap in knowledge, especially in the high Arctic, concerning the pathogens of marine algae, many of which are lower oomycetes.¹⁶

This account presents the methods employed to investigate the macroalgal communities and their associated pathogens in the Cape Hatt region of northern Baffin Island in the Canadian high Arctic during the summer of 2009. The study was diving-based and an outline is given of the risk management for the diving operations and the safety procedures that would have been used if there had been an incident of DCI or other clinically significant diving injury.

Methods

The expedition was staged from Pond Inlet, north Baffin Island from mid-August to early September 2009. Weather permitting, there are either one or two scheduled flights

a day between Pond Inlet and Montreal; the approximate flying distance is 3,700 km. Flight routes differ but there are usually no fewer than three stops on the way with the total transfer time varying between 11 and 32 hours. A field camp was established at Bay 11 in Eclipse Sound, on Cape Hatt (72° 27.8'N 79° 50.4'W¹⁷), which is approximately 85 km to the west of Pond Inlet (Figure 1). Transport between the field camp and Pond Inlet was only by boat; depending on weather and sea ice conditions, transfer time between the two locations was 4–7 hours.

Sampling and recording was undertaken using scuba; there were four divers and the diving adhered to the risk assessment principles of the 1997 UK Diving at Work Regulations.¹⁸ All the diving was shore-based; the divers dived in pairs both attached to a single 100-metre “L” lifeline with a surface tender plus a dive supervisor (Figure 2). The lifeline prevented the divers from drifting too far from the point of entry/egress while maintaining communication between the dive supervisor and both divers in the event of top-side having to terminate a diving operation. All dives were conducted with the availability of a fully independent breathing supply (secondary cylinder and regulator, Figure 2); diving was insured to a maximum operational depth of 15 metres only. Between two and six diving operations were undertaken each day. Dive tasks included collecting seaweed specimens and sediment samples, and taking photographs and video footage (Figure 3).

At all times (24 h per day) during the field component of the expedition, surveillance was maintained in the event of an approach from polar bear (*Ursus maritimus* Phipps). This included times when diving operations were underway; there was always an armed lookout in addition to a tender and dive supervisor in accordance with guidance given in Lang and Sayer (Figure 4).⁴

The emergency evacuation protocol in the case of a diving-related medical problem was based on the probability of not being able to reach a recompression facility within a

Figure 2

Preparing a shore-based diving operation; the divers were tended from the shore using a 100-m “L” lifeline; the support diver (right) acted as an in-water stand-by to the science diver (left); note the use of secondary bailout cylinders (photo courtesy of Olivier Dargent, Nice, France)



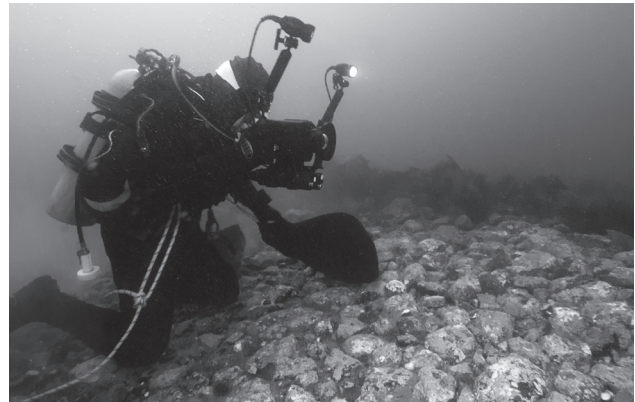
timescale of 24–48 hours. An emergency transfer service was possible from Pond Inlet to Montreal with the capability of transferring divers at a cabin pressure equivalent to an altitude of 214 metres. The health centre at Pond Inlet had sufficient medical and oxygen supplies to stabilize a diver prior to onward transfer; this included a chest drain and Heimlich valve and the capability to carry out urinary catheterisation, if required. The following emergency procedure was developed primarily to cover stabilization in the field plus onward transfer to Pond Inlet.

- On retrieval the diver would be kept supine, warm and dry if possible.
- The diver would immediately be placed on 100% oxygen with enough oxygen for the transfer to Pond Inlet.
- The diver would be given two litres of isotonic fluids initially followed by one litre per hour until they had passed urine; the diver would be given buccal prochlorperazine maleate (*Bucastem*) in the event of any vomiting. The non-steroidal anti-inflammatory drug diclofenac sodium would be administered at 100 mg initially, followed by 50 mg eight hours later.
- A satellite phone would be used to inform the health centre at Pond Inlet while initiating the transfer process on to Montreal.
- An assessment, guided by remote input from medics trained in diving medicine, would be made at Pond Inlet regarding the necessity for onward transfer.

Surface oxygen was available in the field through a Divers Alert Network (DAN) Extended Care Rescue Pack but with additional oxygen bottles capable of delivering a total of at least 360 minutes of oxygen to a single diver; included in the pack were masks that could support breathing and non-breathing divers. An advanced field medical kit was also taken; in addition to an enhanced variety of dressings

Figure 3

Scientific diver performing a video transect of a seaweed-dominated Arctic ecosystem (photo: Martin Sayer, NFSD)

**Figure 4**

A diver filling cylinders using a portable air compressor while also keeping watch for any approaching polar bear (note the rifle); (photo: Martin Sayer, NFSD)



and bandages, the kit included a Guedal airway set (five pieces) and a manual bag resuscitator. Extra drugs carried included: diclofenac (anti-inflammatory/analgesic); codyramol (paracetamol/dihydrocodeine, analgesic); cefradine (antibiotic for chest, skin, urinary infections); ciprofloxacin (antibiotic for ear, chest, skin, urinary infections); prochlorperazine (anti-emetic); loperamide (anti-diarrhoeal); and chlorphenamine (anti-histamine for

allergic reactions). A large volume of rehydration sachets (Dioralyte™) was also taken; all water was boiled, with a volume always available for immediate use.

Results

Exactly 50 diving operations were completed with a total underwater time of 29.7 hours in 12 different locations around the Cape Hatt area. The average maximum operating depth was 10.4 metres' sea water (msw) with an absolute maximum of 15.0 msw. The diving operations generated over 10,000 underwater images, over 20 h of video footage and hundreds of live isolates, herbarium specimens and samples. There were no diving or non-diving incidents; the emergency treatment protocol for an injured diver was not tested.

Discussion

The Arctic could be the defining environment in the continuing debate surrounding climate change. Whereas much discussion and concern has focused on the effects to humankind and top predators, the present study concentrated on developing a baseline against which the potential consequences of change to the ecology of primary producers could be measured. Studies on the identification, classification and cataloguing of the macroalgae and oomycete samples are ongoing and will be reported on elsewhere.

There is no evidence to suggest that diving in cold water presents any elevated risk of developing decompression sickness, although being cold during the decompression phase of a profile (either in the water or immediately following a dive) may not be favourable.⁶ Therefore, in the present study, diving in cold water was not considered to be a major element of the part of the project plan that assessed the risk of DCI. In addition, the shallow nature of the study environments plus the applied insurance limits meant that this expedition carried a very low risk of a diving-related incident. However, the remoteness of the field site was a management concern and there were a number of potential bottlenecks in the transfer route from the dive site to a suitable recompression facility.

The management of mild or marginal DCI in remote locations was the subject of an international workshop in 2004.¹⁹ There were elements to the present study that negated many of the workshop discussions: principally the total lack of any compression chamber within many thousands of kilometres of the study site and the unwillingness to even consider in-water recompression techniques because of limited supplies of oxygen, compressed air and the ambient temperatures. Therefore, the only option was to develop an evacuation plan based on non-recompression therapy.²⁰

The basis of this non-recompression treatment pathway was to make use of the commonly used adjuncts to

recompression therapy, namely breathing 100% oxygen and fluid replacement.²⁰ The use of antiplatelet agents was also advocated, although it was acknowledged that anti-inflammatory inhibitors were likely to provide an additional treatment mechanism. In addition, keeping a patient supine is known to increase nitrogen washout by about 50%. Adjunctive therapies are employed routinely prior to and during emergency recompressions. Although there can be difficulties in determining the true extent of the benefits, normobaric oxygen and aggressive fluid resuscitation would be considered basic first-aid treatments for divers suffering from DCI (with the caveat that aggressive rehydration should be avoided for divers with a suspected isolated cerebral arterial gas embolism).^{21,22} Rehydration therapy using isotonic fluids is not always accepted by the patient and so, if intravenous delivery is not an option, then the management plan should consider employment of buccal prochlorperazine maleate to reduce fluid loss through vomiting.

Traditional management pathways for the treatment of DCI favour seeking rapid recompression. In the present study, the risk assessment process balanced time to treatment against the probability of any incidence of DCI being mild or marginal. That probability was, in turn, managed by limiting depth. There is evidence to suggest that in cases of mild or marginal DCI, no significant disadvantages to long-term clinical outcome are caused by delayed treatment, although the intensity of the symptoms (still mild or marginal) may increase with delay (> 17 h).^{23,24}

DCI was the main health concern for this expedition simply because it was an example of a medical emergency that had never been seen locally by healthcare staff and there was no immediate access to a recompression facility. Polar bear attacks were also a major concern although these are exceedingly rare and the ferocity of polar bears has probably been overemphasized.²⁵ Although firearms were carried at all times, their efficacy in deterring bear attacks has been questioned.²⁶ However, there was good local experience of bear-inflicted wounds and planning for an attack was based on having to treat major trauma in the field with immediate transfer to the Pond Inlet health centre. Hypothermia was also a major concern as any incident would have to have been treated in the field. Diving operations were limited by distance and transfer time back to the field base where re-warming was possible; foil blankets were carried in the field first-aid kit. Other injuries and medical incidents planned for included: diarrhoea/vomiting; slips and trips (elevated risk because of shore-based diving operations); burns (tent fires); infections (particularly ears, chest and urinary) and allergic reactions.

Scientific exploration in remote locations that is dependent on diving should always consider the availability of a suitably equipped research vessel. Where that option is not available, alternative strategies exist within the framework of assessing and managing risk.

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Perceptions amongst Tasmanian recreational scuba divers of the value of a diving medical

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Abstract

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An online survey was offered to recreational divers in Tasmania to ascertain if they have an understanding of how pressure affects their health and if they considered an annual dive medical necessary. A total of 98 recreational divers completed the survey, five of these had never had a dive medical while 74 felt that if they passed their dive medical they do not have any potential illness. Sixty five saw the dive medical as a comprehensive health check. This project provided an insight to Tasmanian recreational divers' understanding of and attitude towards the value of a dive medical.

Key words

Fitness to dive, recreational diving, medicals – diving, survey – diving, computers

Introduction

Recreational diving sits in the collective of swimming/diving reported to the Australian Bureau of Statistics as one of the top five most popular sporting activities in Tasmania.¹ It is challenging to tease out exact participant numbers but there is a noted sustainment of both dive shops and dive schools in Tasmania giving rise to the assumption that recreational diving is holding its appeal. Based on PADI (Professional Association of Diving Instructors) statistics we can also conclude this popularity can be mainly attributed to men in the median demographic of 30 years. Tourism Tasmania lists four recreational dive training schools: two in the north and two in the south, with approximately 100–120 recreational scuba divers trained by each per year. Anecdotally half those qualifying remain in the sport for greater than five years. Reported dropout rates of recreational divers in Western Australia in 2008 suggest this retention rate is high in comparison.²

Before taking a recreational dive course in Tasmania, it is a legal requirement that the candidate undergoes a face-to-face medical examination (AS4005.1) as per Australian Standard AS4005.³ Medical fitness is assessed by a medical practitioner with training in diving medicine.⁴ The medical (AS4005.1) is not designed to be an all-encompassing health check. In recent years its purpose has changed from being the yardstick by which doctors 'police' recreational diving to one of basic health surveillance and advising a level of personal risk mitigation. Indeed, in a 2010 comparative review of personal survey and actual medical outcomes, the face-to-face interview did identify people at risk from diving, but also this component had value beyond making people unfit to dive in that it allows an opportunity for risk mitigation strategies to be offered and discussed.⁵

There are no legal requirements for divers to undergo an

annual or any other interval medical; consequently most recreational divers have a medical at the beginning of their training and never repeat the process. In reviewing 40 diving-related deaths in New Zealand, it was considered that 12 were in divers who should probably have been disqualified from diving on medical grounds.⁶ However, if recreational divers have no obligation to seek a medical it is reasonable to assume that some of these conditions would present in their day-to-day lives.

Method

A concise sample (network sampling) of recreational divers living in Tasmania was recruited for the project in November 2011. Adult, certified recreational divers attending clubs or club-organised dives were invited to complete an anonymous online survey. The questionnaire (available from the author) was advertised via dive group newsletters, online chat rooms and social face-to-face meetings. The researcher was invited to give a presentation of the questionnaire and the research goals at three individual clubs' meetings as well as via podcast and Skype.

The survey questionnaire examines both current perceptions of the participant's health and their attitude towards healthy practices. They were asked to provide some basic demographics and answer questions about their health, their perceptions of personal health and their general attitude to diver health. The online questionnaire took 20 mins to complete. A review of the literature and discussions with a diving medicine physician formed the basis for the original questionnaire design. The questionnaire was refined via a pilot study involving 20 participants and transferred from hardcopy to online on the pilot group's advice. The study was approved by the Human Research Ethics Committee (Tasmania) University of Tasmania, Network REF no: H11783.

Results

Within 5 weeks of starting the project, 98 (75.4%) responses of 130 distributed were received. The demographic data illustrated that the sample was representative of the targeted population. Seventy-one respondents were male, with 39 in the age range 18–28 years. All of those surveyed indicated that they had undertaken some formal dive training. Fifty-five divers had dived more than 15 times during the last year using open-circuit scuba. Twelve of these 55 also dived on hookah (unregulated surface supply via a single hose). A standard scuba second stage is often used as the delivery unit, but light, full-face masks are also used.

When asked about pre-existing medical conditions that may prevent them from diving, 22 of the 98 respondents answered ‘no’ while 63 skipped this question. The remainder gave answers ranging from high blood pressure ($n = 2$), ear injury ($n = 1$), asthma ($n = 2$), diabetes ($n = 1$), achalasia ($n = 1$) and obesity. A similar question was asked later in the survey and phrased slightly differently: “Do you have a medical condition that you think may impact on your diving experience?”. Eleven replied yes and listed ear injury ($n = 1$), asthma ($n = 1$), narrow sinus ($n = 1$), disc L5/S1 pain ($n = 1$), old knee injury ($n = 2$), diabetes ($n = 1$) and achalasia ($n = 1$); these responses correlated with the earlier responses by the same respondents.

Only five had never had a diving medical, 19 had one before their dive course, 19 had one annually, 37 had had their medical more than two years previously and 18 skipped this question (Figure 1). These medicals were performed either by a diving doctor (56) or a general practitioner (42). These numbers include five respondents who had not had a dive medical; it is assumed they indicated a preference should they choose to have a medical. When asked why they did not undergo an annual dive medical, the predominant response was “I don’t feel that my health has changed since my last medical” ($n = 55$). Twelve respondents said they were “too lazy” and 13 stated they “could not afford one”. When asked “If you have a diving medical do you consider this a comprehensive medical check?”, 65 answered yes, 33 said no, while two skipped the question. Respondents validated this by commenting that there were no blood tests ($n = 6$), no test for patent foreman ovale ($n = 1$), no tests for cancer ($n = 4$) and that the “dive medical only checked dive-related stuff”. Several also commented that the medical was designed to check for ‘fitness to dive’ not be a thorough medical exam. The question “If you passed your diving medical would you consider that this means you do not have any potential illness that could compromise your health?” elicited 74 responses. There was no correlation with any particular age group.

When asked about diving illnesses, 80 respondents said that they “knew quite a lot and would be able to render first aid to [themselves] or a buddy” (Figure 2). Three stated that their health could not be compromised by conservative recreational diving. Asked if they had ever had any treatment

Figure 1

Recency of dive medical in years (time) reported by age (years)

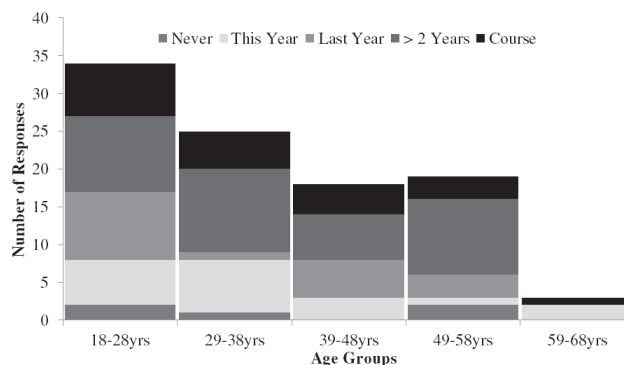
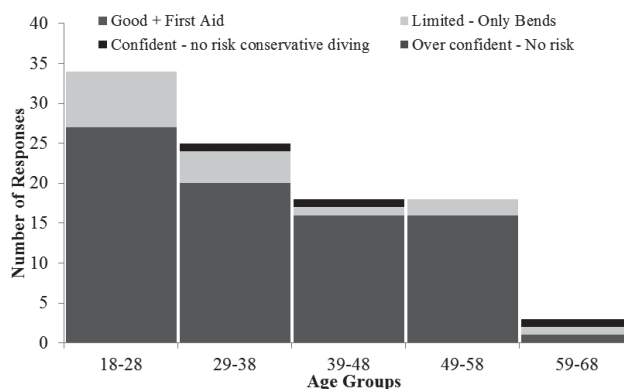


Figure 2

Self-assessed diving illness knowledge is demonstrated to be higher the younger the diver



for a diving-related illness, 78 said no and 20 listed a mixture of ear trouble and decompression illness, (DCI). When asked what might be an injury to make them contact a diving doctor, 73 stated DCI symptoms and the remainder said anything unusual or ear problems after a dive.

When asked if dive medicals should be legislated, 37 said yes and 51 said no. When asked about the time since their last dive medical, there was a cluster of the 18–28-year group who had had a medical either that year ($n = 9$), the previous year ($n = 6$) or associated with a course ($n = 7$). This might be explained by the fact that a large group of university dive club members participated, of whom many would have come recently to the sport. Thirty-seven divers had had their medical over two years previously. This did not correlate with any particular age group, the financial cost ($n = 13$) or lack of time ($n = 4$). Most of the younger divers responded that they felt that “their health had not changed since their last medical so why would they need another?”

Discussion

This survey had a good response rate (75.4%). Many commentators have concerns with the validity of online surveys, specifically the lack of representation of all socio-

economic groups, attributed to variable access to computer facilities.⁷ However, recreational divers are generally 'savvy' with electronic media. The newcomer to recreational diving is often over 18 years of age, financially independent and has a suite of electronic goods with which to access the web, GPS, social media such as *Facebook*, etc.

This project has several important limitations. This dataset was drawn entirely from Tasmania and unintentionally significantly biased to the south of the State. The survey was offered on-line only and there has been no follow up with the cohort to document any changes in opinion.

Despite this, three key themes emerged from the data:

- A dive medical is not a comprehensive health check; respondents felt both 'scans' and 'bloods' should be taken for this to be demonstrated. Several explained that no medical can predict illness, you only find what you are looking for and the dive medical is focused on health issues related to diving, so large collections of ailments associated with anything else are left unassessed or missed.
- The dive medical checks physical aspects of health known to be related to 'fitness to dive', so it can only be assumed you are fit to dive on that day and not completely disease free.
- The third theme is illustrated by the quote; "*Doctors don't know s**t!*" Examples were given of ways that doctors have 'missed' diseases that respondents, friends or family have gone on to develop and suffer from. These were conditions not directly associated with diving fitness, such as rectal cancer.

These themes sit well within the current medical philosophy that a recreational dive medical is an asset to health surveillance rather than a 'big stick' wielded by the physician. In the early 2000s, the move away from the medical practitioner carrying the burden of risk was a significant change in thinking that required some cerebral adjustment by many practitioners in this field.⁸ The predominantly younger generation of recreational divers surveyed for this project are not aware of this medical history nor do they consider the former level of personal health surveillance necessary.

There is also no legislation to prevent recreational divers from diving on the basis of a medical performed when they were younger and fitter. In a report of diving-related deaths in Australian waters in 2008, it was recommended that older divers could reduce their risk by increased health screening.¹⁰ This is supported by a review of the Australian Diver Emergency Service, in which the authors state that "*it has been difficult to persuade recreational divers of the importance of a medical review of fitness beyond their original dive medical*".¹⁰ This situation is clearly echoed in the Tasmanian population, with only 19 of 98 recreational divers surveyed undergoing an annual medical.

Conclusions

This group of recreational divers have provided an insight into their psyche; it is not exclusively the cost or time taken to have a dive medical that they see as prohibitive but a consideration of its necessity. Comprehension of how one's health is affected by pressure may go some way to creating an understanding of why a medical might be of value. Further study to explore recreational divers' understanding and approach to medically managing their health and consequent fitness to dive would be useful.

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British Sub-Aqua Club (BSAC) diving incidents report 2012

Compiled by Brian Cumming, Diving Incidents Advisor

<http://www.bsac.com/core/core_picker/download.asp?id=23411&filetitle=Diving+Incident+Report+2012>

Summary of the 2012 report prepared by Colin Wilson

BSAC started reporting annually on diving incidents in 1980 and these reports continue to be available.^{1,2} Over the years, the amount and quality of the information collected has improved with auditing of the incidents reported to them. Although these data are mainly from reports made by club members, other sources are also used. Information from these reports has allowed BSAC to identify errors and mistakes and the audit cycle has led to changes in training methods to reduce these errors. The reports have been summarised in this journal for a number of years.^{3,4}

As before, the 2012 report covers the United Kingdom (UK) with a few reported incidents involving members while overseas. In total, 314 incidents were reported, the lowest number for a long time and a 15% fall from 2011. This is probably because appalling weather conditions in the summer in the UK curtailed diving. Decompression incident reports were low at 83, with 91 cases of decompression illness (DCI). This downward trend has been running for over 15 years. A caveat, as in previous years, is made that a large number of the cases (51 in total) reported in the “*diver injury/illness*” category are probable DCI cases; this number is less than in previous years. Previous reports have identified the ascent as being a potentially dangerous phase of the dive. This year’s data seem to support the benefit of training directed to prevent ascent errors, with the smallest number (45) recorded since 1999.

As previously mentioned, poor summer weather was a factor affecting the normal monthly distribution, with a mid-season plateau of reports and the loss of the usual June and July peaks. More incidents (13) were recorded at depths greater than 50 metres’ sea water (msw), with three of these being fatalities. Because of the decrease in reports, the involvement of the Coastguard, the Royal National Lifeboat Institute (RNLI) and Search and Rescue (SAR) helicopters was less.

Fatalities

The reduction in incident reports and cases of DCI, however, is not mirrored in the fatalities section, with 17 deaths reported in 2012, above the previous 10-years’ average of 14.7. With such small numbers, attempting to extrapolate a trend may be a step too far. Unfortunately a much greater number (10) than in previous years were BSAC members; we have to go back to 1988 to match this number. Unlike the Australian fatality reports, the quality and depth of information does not make it clear as to what the root causes of these are, though in most cases an educated assessment is made.⁵ More than one cause may be at play when things

start going wrong.

The analysis of the facts show:

- Four cases involved divers suffering a ‘non-diving-related’ medical incident. An additional seven cases were thought to possibly have suffered a ‘medical event’ whilst in the water, though this cannot be substantiated.
- Five cases involved separation of some kind; three had made a conscious decision to separate; the other two separated because of another problem. Separation cannot be deemed the cause of death but it does remove the buddy who may have helped to resolve problems.
- Four cases were using a rebreather; in two of these cases the rebreather did not appear to be a relevant factor; in the other two cases the divers were lost.
- In three cases, the divers were diving alone; in two by choice. The third diver chose to abort during the descent and separated to ascend alone.
- Three cases involved divers diving in a group of three or more.
- Three cases involved dives to depths of 55, 65 and 70 msw; the diver diving to 70 msw had suffered serious trauma before his dive, so depth may not be relevant.
- Two cases involved divers running out of gas, or thinking they had run out of gas.

Previous fatality reports have highlighted that the older diver is over-represented. This is no different in 2012, with an average age of 52.4 years against an average of 38 years for BSAC members as a whole. Eleven of the 17 were 50 years or older, compared with 16% of the diving population (BSAC UK site survey). In relation to the older divers’ health, accurate, honest self-reporting on the medical declaration form is essential. Thereafter, the challenge is still the management of any risks or problems identified.

FROM THE FATALITIES SECTION:

Case 1

“Three divers entered the water for a dive on a wreck at 55 m[sw]. During the descent one of the divers, who was second on the line, had problems with his ears and so paused and ascended slightly. He was passed by the third diver who continued his descent. The diver with ear problems managed to clear them and continued his descent. On the way, he was passed by a diver ascending who[m] he assumed was the third diver in the group but was unsure due to poor visibility. As the two divers reached the wreck they found that the visibility was very poor and decided to abort the dive and ascend. On regaining the boat, they discovered that the diver who had passed them was not their buddy but another diver who had also decided to abort due to the

poor underwater visibility. The pair de-kitted and waited for their buddy to surface. After a time they became concerned as no DSMB [deployable surface marker buoy] was evident but they could see bubbles away from the shotline. One of the divers was preparing to kit up in order to look for the missing third diver when a DSMB surfaced but was not fully inflated. The kitted diver was still concerned and so entered the water and descended the DSMB line. He found the missing buddy but was concerned that the DSMB line was not taut and that his main cylinder was down to 15 bar, although his decompression cylinder had not been used. The diver decided to assist the buddy to the surface and on regaining the boat it was discovered that the missing diver had not completed his safety stops. The diver complained of feeling unwell and asked to lie down. Shortly after, the diver's condition deteriorated, CPR was commenced and the Coastguard was alerted. CPR continued until the arrival of a rescue helicopter which airlifted the unconscious diver to hospital but he did not survive." [Coastguard report]

Case 2

"A diver was preparing to dive on a wreck in a depth of 100 msw using a rebreather. As he moved towards the entry point he slipped and fell to the deck. He was assisted to a bench to recover and the boat was repositioned. Meanwhile his buddy, who had already entered the water, commenced his dive alone. The diver who had fallen then entered the water with another pair and began his descent. At 70 msw this diver decided to abort the dive and made his way back up the shotline. He passed a pair who were decompressing at 50 msw. They reported that he looked distressed and was breathing heavily. A little later they looked up and saw the diver motionless above them with his mouthpiece out of his mouth. They moved up to him and attempted, unsuccessfully, to replace his mouthpiece. They inflated his BCD and sent him to the surface. The diver was recovered into the boat and resuscitation techniques were applied. The Coastguard was alerted and the diver was airlifted to hospital where he was declared dead. It is reported that cause of death was drowning as a result of the effects of serious internal injuries caused by the earlier fall."

Decompression incidents (DCI)

Eighty-three decompression incidents were reported involving 91 cases of DCI. As in previous reports, identifying the cause was difficult in a number of the cases but where identified, and again these may involve a number of causes, they are similar to previous reports as follows:

- 20 repetitive diving;
- 18 rapid ascents;
- 18 diving to depths greater than 30 msw;
- 9 missed decompression stops.

It is again felt that a number of the 'diver injury/illness' reports are probably DCI, though the rate of reporting mirrors that of previous years.

FROM THE DCI SECTION:

Case 3

"A diver conducted two dives a day over a weekend with dives on day one no deeper than 10 msw. On day two she dived to 18 m and then, after a surface interval of 2 hours 15 min, she dived to a maximum depth of 20 msw. During both dives on the second day, the diver experienced buoyancy problems with her drysuit, feeling that air had become trapped. She had difficulty maintaining a 6 msw safety stop on the first dive, rising to 2 msw and then fighting to get back to 6 msw. On the second dive she could not control the stop at all and ascended direct to the surface and remained there. After a surface interval of around 5 hours, the diver began a car journey home. An hour later she began to feel unwell with a headache and fatigue which both gradually increased until she fell asleep. On returning home, the diver took pain relief and went to bed but had a restless night. The following morning the diver noticed a purple rash in clusters on her chest, neck and back; she still had the headache and felt tired and lightheaded. The diver discussed it with her husband but felt uncomfortable phoning a recompression chamber. The diver was eventually persuaded to call the chamber and after describing her symptoms was advised to attend straight away. The diver attended a recompression chamber and was recompressed on a Royal Navy Table 62 with extensions and received a follow-up treatment the next day."

Case 4

"A diver and his buddy completed a dive without incident to a maximum depth of 25 msw and a total duration of 46 min with a 1 min stop at 6 msw. Approximately 4 hours after the dive the diver went to work and then started to experience a slight pain in his knees and a tingling sensation around his face. The diver contacted his buddy for advice regarding the symptoms and was advised to call a duty doctor at a recompression chamber who suggested that he attend for further examination. The examination was inconclusive but the diver was advised to undergo a precautionary recompression treatment for 4 hours. The diver had this recompression treatment and was discharged home the following day. His buddy did not experience any symptoms. The diver had suffered a serious head injury some months previously that had resulted in being unconscious for approximately 4 hours but had been advised at A&E that it should not prevent him diving. The diver has sought further medical advice and been advised not to dive for nine months."

The low number of DCI cases in this report needs to be viewed in light of the overall reduction in dive incident reports and further years' reports may put this better into perspective. Similarly to previous reports, it demonstrates the common failures occurring and helps direct education and learning. Thanks go to Brian Cumming and his team at BSAC for collating this report, but we must also acknowledge those who have honestly reported their failures and misdemeanours.

References

- 1 *BSAC diving incident report archive 1980 to 1999*. Accessed 15 October 2013. Available from <http://www.bsac.com/page.asp?section=2619§ionTitle=Diving+Incident+Report+Archive>
- 2 *BSAC annual diving incident report 2000 to 2012*. Accessed 15 October 2013. Available from: <http://www.bsac.com/page.asp?section=1038>
- 3 Wilson CM. British Sub-Aqua Club (BSAC) diving incident report 2007. *Diving Hyperb Med*. 2008;38:165-6.
- 4 Wilson CM. British Sub-Aqua Club (BSAC) diving incident report 2011. *Diving Hyperb Med*. 2012;42:234-5.
- 5 Lippmann J, Walker D, Lawrence CL, Wodack T, Fock A, Jamieson S, et al. Provisional report on diving-related fatalities in Australian waters 2008. *Diving Hyperb Med*. 2012;43:16-34.

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Key words

Recreational diving, accidents, diving deaths, abstracts, case reports

Letters to the Editor

Despite animal studies, HBOT is the treatment of choice for cerebral gas embolism

Following our case report on retrograde cerebral venous gas embolism (CVGE),¹ we wish to draw the attention of readers of this journal to a paper in *Critical Care Medicine* by Weenink et al reporting that severe cerebral arterial gas embolism (CAGE) is a lethal injury in a swine model, not salvageable with hyperbaric oxygen.² This we already know from managing some very unfortunate patients.

Weenink and co-workers have done important research into CAGE, hyperbaric oxygen treatment (HBOT) and animal models.^{2,3} Unfortunately, the less well informed reader may find their title: "*Hyperbaric oxygen does not improve cerebral function when started 2 or 4 hours after cerebral arterial gas embolism in swine*" enough to abandon the idea of HBOT.² The more astute reader, we hope, will concur with us that this study has many shortcomings: the injuries inflicted were excessive, only one HBOT session was administered, no clinical follow up was possible, no imaging was done, the inflammatory process was not quantified, and no histology was undertaken.²

Administering smaller quantities of air and then correlating the effect with imaging to compare such moderately

severe cases of CAGE with retrograde cerebral venous gas embolism (CVGE) of similar quantity will be extremely useful.^{4,5} Comparing the duration of gas present in the cerebral arterial versus cerebral venous system will shed some important light on this newly recognised phenomenon of CVGE. Comparing imaging, outcomes, response to HBOT and histology will also be important.

Their assertion that clinical outcome variables are of vital importance is correct, but we disagree that studying animal models is the only way forward. With the human brain being such a complex structure with complex functions, we do not think outcomes can be accurately assessed in an animal model. With the recognition of CVGE as a separate entity we now have to look at this 'new' category, and need the co-operation of all clinicians dealing with air embolism to report their cases to our journals and also to the air embolism registry that has been established recently in the United Kingdom (<http://www.gasembolism.org.uk>).

We will be collecting extensive, anonymous data from all cases with CVGE and CAGE notified to the registry. Hopefully we may be able to see if patients with CVGE may 'get away' without HBOT in the recommended 6–7 hour time frame, which is often a difficult target if a hyperbaric unit is not available on site. Assessing the possibility of fewer HBOT sessions or any benefit at all for cases with CVGE will be important.^{1,6}

References

- 1 Bothma PA, Brodbeck AE, Smith BA. Cerebral venous air embolism treated with hyperbaric oxygen: a case report. *Diving Hyperb Med*. 2012;42:101-3.
- 2 Weenink RP, Hollmann MW, Vrijdag XC, Van Lienden KP, De Boo DW, Stevens MF, et al. Hyperbaric oxygen does not improve cerebral function when started 2 or 4 hours after cerebral arterial gas embolism in swine. *Crit Care Med*. 2013;41:1719-27.
- 3 Weenink RP, Hollmann MW, van Hulst RA. Animal models of cerebral arterial gas embolism. *J Neurosci Methods*. 2012;205:233-45.
- 4 Schlimp CJ, Loimer T, Rieger M, Lederer W, Schmidts MB. The potential of venous air embolism ascending retrograde to the brain. *J Forensic Sci*. 2005;50:906-9.
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- 6 Lai D, Jovin TG, Jadhav AP. Cortical vein air emboli with gyriform infarcts. *JAMA Neurol*. 2013;70:939-40.

Pieter A Bothma, Consultant in anaesthesia, London Hyperbaric Unit, James Paget University Hospital, Gorleston, United Kingdom. Robin EA Heij, Registrar in anaesthesia, East of England Hyperbaric Unit, Addenbrooke's Hospital, Cambridge, UK.

E-mail: <pabothma@gmail.com>

Key words

Cerebral arterial gas embolism (CAGE), venous gas embolism, arterial gas embolism, clinical audit, letters (to the Editor)

New SPUMS Education Officer needed

I will be resigning as the SPUMS Education Officer as of the May 2014 SPUMS AGM. This letter is to broadcast this opening to as wide an audience as possible. I realise we are a small group and everyone is busy with their varied professional and personal commitments. However, we need to pass the baton around and share the load, so we can advance the speciality!

Please contact the Secretary: <secretary@spums.org.au> or myself for further details if you are interested in assuming

this very important role within the Society. Candidates must be full members of SPUMS and hold the SPUMS Diploma.

Thank you for your enthusiasm and support.

*Associate Professor David Smart, Medical Director,
Department of Diving and Hyperbaric Medicine, Hobart
Hospital, Hobart, Tasmania.*

E-mail: <david.smart@dhhs.tas.gov.au>

Key words

Medical society, research, letters (to the Editor)

The database of randomised controlled trials in hyperbaric medicine maintained by Michael Bennett and his colleagues at the Prince of Wales Hospital Diving and Hyperbaric Medicine Unit, Sydney is at:
<<http://hboevidence.unsw.wikispaces.net/>>

Assistance from interested physicians in preparing critical appraisals is welcomed, indeed needed, as there is a considerable backlog. Guidance on completing a CAT is provided. Contact Associate Professor Michael Bennett: <M.Bennett@unsw.edu.au>

Advertising in *Diving and Hyperbaric Medicine*

Commercial advertising is welcomed within the pages of *Diving and Hyperbaric Medicine*. Companies and organisations within the diving, hyperbaric medicine and wound-care communities who might wish to advertise their equipment and services are welcome.

The advertising policy of the parent societies – EUBS and SPUMS – appears on the journal website: <www.dhmjournal.com>

Details of advertising rates and formatting requirements are available on request from:

E-mail: <editorialassist@dhmjournal.com>

Fax: +64-(0)3-329-6810

The
Diving and Hyperbaric Medicine
journal website is at
<www.dhmjournal.com>

Continuing professional development

Diabetes and diving

CME activity 2013/4

Michael Bennett

Accreditation statement

INTENDED AUDIENCE

The intended audience consists of all physicians subscribing to *Diving and Hyperbaric Medicine* (DHM), including anaesthetists and other specialists who are members of the Australia and New Zealand College of Anaesthetists (ANZCA) Diving and Hyperbaric Medicine Special Interest Group (DHM SIG). However, all subscribers to DHM may apply to their respective CPD programme coordinator or specialty college for approval of participation.

This activity, published in association with DHM, is accredited by the ANZCA Continuing Professional Development Programme for members of the ANZCA DHM SIG under Learning Projects: Category 2 / Level 2: 2 credits per hour.

OBJECTIVES

The questions are designed to affirm the participants' knowledge of the topics covered, and participants should be able to evaluate the appropriateness of the clinical information as it applies to the provision of patient care.

FACULTY DISCLOSURE

Authors of these activities are required to disclose activities and relationships that, if known to others, might be viewed as a conflict of interest. Any such author disclosures will be published with each relevant CPD activity.

DO I HAVE TO PAY?

All activities are free to subscribers.

Key words

MOPS (maintenance of professional standards), diabetes, medical conditions and problems, fitness to dive

Recommended background reading

Practitioners are referred to the following background references and reading.

- 1 Kuzuya T, Matsuda A. Classification of diabetes on the basis of etiologies versus degree of insulin deficiency. *Diabetes Care*. 1997;20:219-20.
- 2 The South Pacific Underwater Medicine Society. *Guidelines on medical risk assessment for recreational diving 2010*. Available at: [http://www.spums.org.au/public-file-download/full-spums-medical_\(last accessed 14 November 2013\)](http://www.spums.org.au/public-file-download/full-spums-medical_(last%20accessed%2014%20November%202013).).
- 3 Retnakaran R, Zinman B. Type I diabetes, hyperglycaemia and the heart. *Lancet*. 2008;371:1790-9.
- 4 Edge CJ; St Leger Dowse M; Bryson PJ. Scuba diving with diabetes mellitus – the UK experience 1991-2001. *Undersea Hyperb Med*. 2005;32:27-37. Available at: <http://archive.rubicon-foundation.org/xmlui/handle/123456789/4036?show=full>

How to answer the questions

Please answer all responses (A to E) as True or False.

Answers should be posted by e-mail to the nominated CPD co-ordinator.

For ANZCA DHM SIG and other SPUMS members, this will be Michael Bennett, **E-mail:** <m.bennett@unsw.edu.au>. For EUBS members for this CPD issue this will be Lesley Blogg, **E-mail:** <lesley.blogg@eubs.org>.

On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect. A correct response rate of 80% or more is required to successfully undertake the activity. Each task will expire within 24 months of its publication to ensure that additional, more recent data have not superseded the activity.

Question 1. Regarding insulin:

- A. Insulin has generally anabolic effects on a number of organs.
- B. Insulin causes cells in the liver to take up glucose and convert it to glycogen.
- C. Insulin promotes the use of fat as an energy source.
- D. Insulin blocks lipolysis and the production of fatty acids.
- E. Insulin has a role in amino acid uptake by cells.

Question 2. Regarding the causes and classification of diabetes mellitus:

- A. Type I diabetes is often an autoimmune phenomenon that leads to the destruction of islet cells.
- B. Type II diabetes represents about 60% of cases of diabetes mellitus.
- C. There is a strong association between obesity and the development of Type II diabetes.
- D. Both the incidence and prevalence of diabetes is rising on a global scale.
- E. Gestational diabetes may precede the development of Type I diabetes.

Question 3. Aspects of diabetes that may have implications for diving fitness include:

- A. Peripheral neuropathy may confuse a diagnosis of decompression illness.
- B. Hypoglycaemic unawareness is uncommon in Type I diabetics with tight control.
- C. Hypoglycaemia may be precipitated by cold and exercise.
- D. There is a high prevalence of coronary arterial disease in the diabetic population.
- E. Autonomic neuropathy suggests a blunting of adrenaline release in response to hypoglycaemia.

Question 4. Regarding Type II diabetes mellitus and diving:

- A. SPUMS recommends that diabetics over the age of 40 years have annual exercise electrocardiography.
- B. Autonomic neuropathy impairs the sharpened Romberg test and therefore this assessment is a poor diagnostic tool in DM.
- C. Individuals with a higher glycosylated haemoglobin percentage are at higher risk of vascular complications.
- D. Retinopathy may impair an individual's ability to read their gauges.
- E. According to SPUMS, Type II diabetics may be able to learn to dive three months after starting oral hypoglycaemic agents if blood sugar is well controlled.

Question 5. SPUMS guidelines allow people with diabetes to undertake scuba diving if the following criteria are met:

- A. The candidate is over 16 years of age.
- B. At least one year has passed since the initiation of insulin therapy.
- C. The HbA1c is < 9.1 mmol L⁻¹.
- D. They do not inform other divers they are with of the diagnosis, so they do not become apprehensive.
- E. Training is undertaken in the context of a specifically designed course for diabetic divers.



Notices and news

EUBS news now on the website

EUBS notices and news, such as the minutes of the most recent Executive Committee meeting and the 2013 General Assembly held during the Tricontinental meeting on Reunion Island, can now be found, along with all other information about the Society, on the website

<www.eubs.org>. In order to increase space for original research and educational articles and to minimise the rising costs of publishing *Diving and Hyperbaric Medicine*, the decision has been made to reduce the amount of society business appearing within these pages in future.

European Editor retiring, a successor is needed

EUBS announces with sadness that the European Editor, Peter Müller, will resign at the EUBS 2014 ASM next September. A search has been established to appoint a new European Editor by May 2014, so that (s)he has time to overlap with Peter.

Candidates for the position should send a brief application and their full curriculum vitae, in electronic format, to the Honorary Secretary, Joerg Schmutz at:
E-mail: <joerg.schmutz@eubs.org>.

Zetterström Award – Réunion2013

The Zetterström Award, Réunion2013 was given for the poster:

D Cialoni, M Pieri and A Marroni. *Flying after diving: in-flight echocardiographic study after a week of intensive recreational diving.*

Musimu award – Réunion2013

The Musimu Award, Réunion2013 was given for the poster:

M Rozloznik, C Balestra and M Dutschmann. *Comparative analysis of expression and plasticity of the diving bradycardia triggered by repetitive face immersions in inspiration and expiration.*



First announcement and call for abstracts

Dates: 24–27 September 2014

Venue: Wiesbaden, Germany

The 40th EUBS Annual Scientific Meeting will be held in conjunction with the 2014 congress of the German Society for Diving and Hyperbaric Medicine (GTÜeM) with Peter Müller serving as the Secretary General for the meetings.

Organising Committee

Peter Müller (Secretary General), Peter Germonpré (EUBS), Karin Hasmler (EUBS/GTÜeM), Michael Kemmerer (EUBS/VDD/Wiesbaden), Dirk Michaelis (EUBS/GTÜeM/Wiesbaden)

Scientific Committee

Costantino Balestra (EUBS), Lesley Blogg (EUBS), Bjorn Jüttner (EUBS/GTÜeM), Claus-Martin Muth (EUBS/GTÜeM), Lars Perlik (Wiesbaden), Tim Piepho (GTÜeM), Christian Weber (Frankfurt), Christian Werner (Mainz)

Main topics

- Invited lectures: marine biology; carbon monoxide toxicity; stem cells and HBOT
- Diving medicine: physiology; decompression theory; treatment
- HBO medicine: physiology; treatment; technical and safety aspects
- Pro/Con debate
- GTÜeM Guideline: Treatment of diving accidents

The meeting format will be the usual EUBS style, with invited keynote lectures, presentations of free papers (oral and posters) and an industry exhibition.

Call for abstracts

Abstracts for oral and poster presentations should be submitted electronically via <www.eubs2014.org> The Organising Committee intends to publish all accepted abstracts in a conference book and encourages all authors to submit full papers for consideration in *Diving and Hyperbaric Medicine*.

Preliminary timetable

Registration is open via the website: <www.eubs2014.org>

30 April: Deadline for submission of abstracts

01 May: End of early bird registration period

15 July: Notification of accepted abstracts

Language: The official language for all scientific sessions and the International DAN Diver's Day will be English. The language for the the GTÜeM Guideline "*Treatment of Diving Accidents*" will be German.

For further information and hotel bookings see: <www.eubs2014.org>

Conference Secretariat: Phone: +49-(0)611-847-27-170

Fax: +49-(0)611-847-27-179

E-mail: <info@eubs2014.org>



website is at
<www.eubs.org>



website is at
<www.spums.org.au>

Members are encouraged to log in and to keep their personal details up to date

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Notices and news

SPUMS news now on the website

SPUMS notices and news, such as the minutes of the November 2012 Executive Committee meeting and the 2013 Annual General Meeting held during the Tricontinental meeting on Réunion Island, including the officers' and financial reports, can now be found, along with all other information about the Society, on the website <www.spums.org.au>. In order to increase space for original research and educational articles and to minimise the rising costs of publishing *Diving and Hyperbaric Medicine*, the decision has been made by the Executive to reduce the amount of society business appearing within these pages in future and for this to be accessible for members on the website.

SPUMS Annual Scientific Meeting 2014

Venue: Alila Manggis Resort, Bali

Dates: 18–25 May 2014

Themes:

Patent foramen ovale (PFO); immersion pulmonary oedema; the older diver

Keynote speaker:

Peter Wilmschurst, Cardiologist, UK

Submission of abstracts:

There are only limited time slots remaining for presenting a paper so please submit abstracts ASAP!

Registration:

Registration, resort bookings, diving details and more information are on the SPUMS website:

<www.spums.org.au>

Register now – The conference resort is now full and the meeting must be capped at 80 registrants.

Resort facilities can be viewed at: <<http://www.alilahotels.com/manggis>>

There are extensive alternative accommodation options nearby.

A wait list for Alila will be started in case of existing bookings being cancelled.

SPUMS ASM 2014 Convenor: Neil Banham

E-mail: <N.Banham@health.wa.gov.au>

SPUMS Diploma in Diving and Hyperbaric Medicine

The full requirements for the SPUMS Diploma and all additional information can be found on the society website: <www.spums.org.au>.

The Education Officer's report of July 2013 may also be found on the website. This contains details of candidates who have registered projects for the Diploma in the past three years and the stage at which each of these has reached.

All enquiries and applications should be sent to:

Associate Professor David Smart

GPO Box 463, Hobart, Tasmania 7001

E-mail: <david.smart@dhhs.tas.gov.au>

Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists

Eligible candidates are invited to present for the examination for the Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists.

All details are available on the ANZCA website at:

<<http://anzca.edu.au/edutrainning/DHM/index.htm>>

Suzu Szekely, FANZCA, Chair, ANZCA/ASA Special Interest Group in diving and hyperbaric medicine.

E-mail: <Suzu.Szekely@health.sa.gov.au>

SPUMS and Facebook

Remember to 'like' SPUMS at:

<<http://www.facebook.com/pages/SPUMS-South-Pacific-Underwater-Medicine-Society/221855494509119>>

The ANZ Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine 2014

Dates: 24 February–07 March

Venue: Prince of Wales Hospital, Sydney, Australia

Course content includes:

- History of hyperbaric oxygen
- Physics and physiology of compression
- Accepted indications of hyperbaric oxygen
- Wound assessment including transcutaneous oximetry
- Visit to HMAS Penguin
- Visit to the NSW Water Police
- Marine envenomation
- Practical sessions including assessment of fitness to dive

Approved as a CPD Learning Project by ANZCA: Cat 2, Level 2 – 2 credits per h (approval no. 1191).

Contact for information:

Ms Gabrielle Janik, Course Administrator

Phone: +61-(0)2-9382-3880

Fax: +61-(0)2-9382-3882

E-mail: <Gabrielle.Janik@sesiahs.health.nsw.gov.au>

Hyperbaric Oxygen, Karolinska

Welcome to: <<http://www.hyperbaricoxygen.se/>>.

This site, supported by the Karolinska University Hospital, Stockholm, Sweden, offers publications and free, high-quality video lectures from leading authorities and principal investigators in the field of hyperbaric medicine.

You need to register to obtain a password via e-mail. Once registered, watch the lectures online, or download them to your iPhone or computer for later viewing.

We offer video lectures from:

- The 5th Karolinska PG course in clinical hyperbaric oxygen therapy, 07 May 2009.
- The European Committee for Hyperbaric Medicine “Oxygen and infection” Conference, 08–09 May 2009.
- The 17th International Congress on Hyperbaric Medicine, Cape Town, 17–18 March 2011.

Also available is the 2011 Stockholm County Council report: *Treatment with hyperbaric oxygen (HBO) at the Karolinska University Hospital*.

For further information contact:

Folke Lind, MD PhD

E-mail: <folke.lind@karolinska.se>

Website: <www.hyperbaricoxygen.se/>

Royal Adelaide Hospital Hyperbaric Medicine Unit Courses 2014

Medical Officers' Course

Part 1: 01–05 December (Lectures)

Part 2: 08–12 December

DMT Full Courses

24 February–14 March

06–24 October

DMT Refresher Courses

28 April–09 May

22 Sept–03 Oct

All enquiries to:

Lorna Mirabelli, Course Administrator

Phone: +61-(0)8-8222-5116

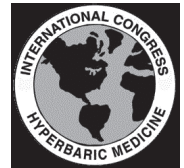
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18th International Congress on Hyperbaric Medicine

03–06 December 2014

Buenos Aires, Argentina



The ICHM is a worldwide organization for physicians and scientists interested in diving and hyperbaric medicine. The organization has minimal formal structure and is entirely dedicated to hosting an international scientific congress every three years.

ICHM Committee (2011–2014):

President: Prof Dr Jorge B Pisarello (Argentina)

Executive Director: Dr Alessandro Marroni (Italy)

Secretary: Assoc Prof Michael Bennett (Australia)

Registration: Online registration opens 30 November 2013

Website:

<<http://ichm.drupalgardens.com/content/what-ichm-0>>



DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia

E-mail:
<deswill@dingley.net>

Website:
<www.classicdiver.org/>

Scott Haldane Foundation

The Scott Haldane Foundation (SHF) is an international institute for the training and education of diving medical examiners and diving medicine physicians.



It was founded in 1976 to spread diving medical know-how among physicians and to provide support for those wishing to attend international diving medical meetings. In those old days, SHF also published a booklet about diving medicine and issued a magazine for a short period of time.

After hibernating for a while, in 2003 a revived SHF started to develop regular courses in diving and hyperbaric medicine (DHM). In the past decade, over 2,000 physicians have participated in SHF courses. Though SHF's origins lie in The Netherlands, courses have been run in more than 20 countries, from Honduras to Indonesia and Tanzania to Belgium. The SHF faculties consist of world-renowned specialists in diving medicine, from Australasia, the USA and Europe.

The SHF courses also laid the fundamentals for a certification system in The Netherlands for diving medical examiners and diving physicians. Since 2011, SHF has been certified by the European College of Baromedicine as providing internationally accredited courses in DHM. All courses meet EDTC/ECHM guidelines, leading to certification as Medical Examiner of Divers (level 1) or Diving Medicine Physician

Capita Selecta Duikgeneeskunde Medication and diving

Date: 08 February 2014

This course is intended to provide an insight into drug interactions, both good and bad, in relationship to diving.

For more information see websites: <diversearch.prg> (EN) or <duikresearch.org> (NL)

Undersea and Hyperbaric Medicine Society Annual Scientific Meeting 2014

Dates: 19–21 June

Venue: Hyatt Regency St Louis at the Arch

For full information go to: <www.uhms.org>

(DMP) (level 2d). The programmes have a modular structure allowing students to attain certification in several steps. Thanks to this modular design and the varied topics, the courses also meet CME requirements.

As well as the diving medicine courses, SHF also organizes two internships for the DMP certification. The first is in hyperbaric medicine and the second focuses on the various diving systems used in both recreational and professional diving. SHF is also involved in the organisation of international conferences in DHM, most recently the very successful first Tricontinental conference on Réunion Island in September 2013 for the EUBS, SPUMS and SAUHMA.

To learn more about the SHF courses and activities, please visit: <www.scotthaldane.org>.

Provisional course calendar 2014

25 January: Refresher course Fitness to dive. AMC, Amsterdam, The Netherlands

29 March–04 April: Basic course (med. exam. of divers) Part 1. Loosdrecht, NL

05, 11 & 12 April: Basic course (med. exam. of divers) Part 2. AMC, Amsterdam, NL

17–24 May: Basic course (med. exam. of divers) Part 2. Al Sawadi, Oman

October: (dates tba): Refresher course. AMC, Amsterdam

08–15 November Basic course (med. exam. of divers) Part 1. tba, tropical location

15–22 November: 22nd In-depth course Diving medicine. tba, tropical location

22–29 November: 22nd In-depth course Diving medicine. tba, tropical location

German Society for Diving and Hyperbaric Medicine

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by the German Society for Diving and Hyperbaric Medicine (GTÜeM) according to EDTC/ECHM curricula, can be found on the website: <http://www.gtuem.org/212/Kurse/_Termin/Kurse.html>

DAN Europe

DAN Europe has a fresh, multilingual selection of recent news, articles and events featuring DAN and its staff.

Go to the website: <<http://www.daneurope.org/web/guest/>>

Instructions to authors

The 'short' version of the *Instructions to Authors* will no longer be printed in each issue of the Journal. Please refer to the *Diving and Hyperbaric Medicine* website: <www.dhmjournal.com> for a downloadable pdf of the full instructions (revised July 2011). A revision will be published in the March 2014 issue and thereafter will be available on the website.

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA

1800-088200 (in Australia, toll-free)
+61-8-8212-9242 (International)

SOUTHERN AFRICA

0800-020111 (in South Africa, toll-free)
+27-10-209-8112 (International, call collect)

NEW ZEALAND

0800-4DES-111 (in New Zealand, toll-free)
+64-9-445-8454 (International)

EUROPE

+39-6-4211-8685 (24-hour hotline)

ASIA

+10-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

UNITED KINGDOM

+44-7740-251-635

USA

+1-919-684-9111

The DES numbers (except UK) are generously supported by DAN

DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. All information is treated confidentially with regard to identifying details when utilised in reports on fatal and non-fatal cases. Such reports may be used by interested parties to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence unless otherwise agreed) to:

DAN Research
Divers Alert Network Asia Pacific
PO Box 384, Ashburton VIC 3147, Australia
Enquiries to: <research@danasiapacific.org>

DAN Asia-Pacific NON-FATAL DIVING INCIDENTS REPORTING (NFDIR)

NFDIR is an ongoing study of diving incidents, formerly known as the Diving Incident Monitoring Study (DIMS). 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.

The NFDIR reporting form can be accessed on line at the DAN AP website:
<www.danasiapacific.org/main/accident/nfdir.php>

DISCLAIMER

All opinions expressed in this publication are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policies or views of SPUMS or EUBS or the Editor.

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