THE ANATOMY OF DEATH IN TWO DIVERS

Chris Acott

Key Words

Case report, cerebral arterial gas embolism, deaths, drowning, pulmonary barotrauma.

Introduction

Between New Year's Day and this conference in May 2001, two divers have died in South Australia. We have a well informed Coroner in South Australia who insists that in any deaths involving divers, either I or a doctor from the Hyperbaric Unit, attends the post-mortem and directs the pathologist on what to look for.

It usually assumed, and has been for many years, that novice divers probably die of cerebral arterial gas emboli (CAGE) following panic or distraction causing breath holding during ascent. More experienced divers die of many other things as well as CAGE due to pulmonary barotrauma. Some divers develop CAGE from bubbles passing through a patent foramen ovale after a fairly strenuous dive.

What to do at a diving death

In any diving death it is necessary to make sure that the equipment is impounded. After the remaining air pressure has been be recorded, the cylinder(s) should be turned off and the number of turns required recorded. Nobody at the scene should touch the equipment after that because it will be needed for analysis and checking for function later. If possible, photograph the body as it is brought out of the water or lying on the beach. Photograph the body with the equipment on. I know it sounds very morbid but it is amazing what you can find out from a photograph. It is important to photograph each individual piece of equipment, particularly the contents gauge to register how much air was in the cylinder(s) at the end of the dive. The most important thing is to make sure nobody interferes with the equipment.

Confusing factors

One of the problems with diving deaths is that you may actually get wrong information about divers or the dive even from a relative. One really has to follow up and find out exactly what went wrong.

Case 1

THE INCIDENT

This unfortunate 45 year old female diver surfaced and signalled to the boat. However there was no response to the boat's return signal and the diver was seen to submerge. The dive master surfaced and was pointed to where the diver had submerged, swam over and found the diver in about 5 m with the regulator out of her mouth. The regulator was not replaced during the rescue and the ascent to the surface. When the diver was hauled into the dive boat they were unable to resuscitate her. There was a doctor and nurse on board, so it is likely that the resuscitation attempts were quite good. She was pronounced dead at the local hospital. I was contacted soon after that by the local police. I told the them to impound the diver's equipment. There was between 50 to 80 bar still left in the cylinder.

I contacted the local hospital, spoke to the duty GP and asked him to organise a chest X-ray of the body supine and sitting if at all possible. That sounds very morbid but one is looking at a sudden, unexpected death and it is important to see if there is air in the heart. If people object to sitting the body up, it can be rolled onto a side which may show fluid levels in the heart.

THE ORIGINAL HISTORY

The dive was to 18 to 20 m for about 25 minutes, so a lot more air was used than one would expect, to leave only about 50 to 80 bar in the cylinder. The buoyancy compensation device (BCD) was uninflated, there was no evidence of rapid ascent precipitated by the BCD. There were no apparent problems during the drive other than separation during the dive and the diver appeared fairly comfortable. She was supposed to be an experienced cave diver. She was supposed to have had a recent diving medical. Her son thought she was doing a refresher course in diving. She had a recent hospitalisation for a laparoscopic appendicectomy and the son thought that she had no medical problems before this.

BEFORE THE POST-MORTEM

I thought cerebral aterial gas embolism was probably unlikely. She was an experienced cave diver so should have good buoyancy control. I thought that it might be a sudden death from a myocardial infarction, sub-arachnoid haemorrhage or intra-cerebral bleed. The X-rays showed massive pulmonary oedema.

THE POST-MORTEM

She was a fairly large lady, who had bitten her tongue on her right side. She had marks on her chest consistent with CPR. I checked her ears for signs of ear barotrauma as even a Valsalva during the ascent to try and clear the ears can precipitate pulmonary barotrauma and CAGE. There was no sign of any cerebral vascular event.

When the chest cavity was opened we found there were pleural adhesions in the right middle lobe and right lower lobe. She did not have a patent foramen ovale. There

was vomit in the trachea. There was no sign of a recent myocardial infarction and the pathologist said, "Oh, the lady drowned". There was no air seen in the heart. No pneumothorax or mediastinal gas of any description was present.

But why did she drown? That is what we wanted to know.

CAUSE OF DEATH

Looking at the unexpected findings, the bitten tongue and the pleural adhesions I postulated the cause of death was CAGE. I thought that the bitten tongue was consistent with a convulsion underwater, otherwise why would her tongue have been bitten? The pleural adhesions had caused a local change in lung compliance and hence she ended up with pulmonary barotrauma and gas embolism.

LATER HISTORY

Later it came out that her son had got everything completely wrong. She was not an experienced diver. In fact this was only her 8th ever dive. I have no idea where he got the information that his mother was a cave diver. This was her first dive following a laparoscopic appendicectomy which was performed at the Flinders Medical Centre. Her medical history from the Flinders Medical Centre said that she was an asthmatic. She had suffered a pneumothorax in about 1996. She had had frequent chest infections throughout her life as well as frequent bouts of abdominal pain from endometriosis. The pathologist's report of the appendix removed laparoscopically was that the appendix was normal and the pain was probably another bout of endometriosis. The chest X-ray done on her in 1997 showed an ill defined increase in density in the right middle zone. It showed definite focal consolidation in the lateral films, however there was an area of increased capacity anteriorly, probably a pleural thickening and a parietal lesion, which is what we found in the post-mortem.

Her diving medical was quite interesting. She answered "No" to chest problems in the past but there was a question mark by this question, and who put that there is yet to be determined, and probably will be determined in the Coroner's Court. The diving medical was performed by a general practitioner in Adelaide. He is not known to me and I think I probably know everybody in Adelaide who has the proper training and does diving medicals.

Case 2

THE INCIDENT

A month later a diver surfaced with his buddy and then died on the surface.

THE ORIGINAL HISTORY

Again he was reported as being an experienced diver. He was known to be on anti-hypertensive treatment and the death was recorded in the paper as being from a heart attack on the surface. The ascent was controlled. He died on the surface.

The dive was fairly innocuous. It was the second dive of the day using fresh cylinders. Two or three hours earlier they had done a similar dive. The dive team consisted of the victim, the victim's wife and a friend who was leading the dive. After 20 minutes at 10 m the victim had 50 bar in his cylinder. The others had between 100 and 150 bar. The friend and the victim ascended leaving the wife on the bottom. The victim was told to inflate his BCD and float while his friend went down to bring his wife to the surface because they were both very novice divers. He was alright on the surface. He spoke to his friend. The friend then descended, collected the wife at 10 m and brought her to the surface. This did not take long. When they returned to the surface the friend noticed that the victim was floating or sunning himself on his back. When he called out to him to come back to the boat there was no reply. So the friend swam over to the victim and found his buoyancy jacket was over inflated. The victim was unresponsive and he had vomit dripping from the side of his mouth. The friend did not know whether he was breathing or not. Resuscitation was unsuccessful.

BEFORE THE POST-MORTEM

I was able to get a chest X-ray done but it was not done in the sitting position but on the side. The victim was very overweight and that was one of the reasons why they could not sit him up. He was huge. There was no pneumothorax on the chest X-ray. There were fluid levels and air in the heart.

POST-MORTEM

Inspection of his mouth showed that he also had bitten his tongue on the right side, just like the other diver. There were pleural adhesions in his left upper chest. His coronary arteries, surprising enough, were clear. The pathologist said "one could drive a truck through them, so we are probably not dealing with a heart attack". There was no evidence of a cerebral vascular event. However there was air in the heart.

CAUSE OF DEATH

There is no doubt in my mind that this man died from an embolic event, CAGE, leading to drowning.

LATER HISTORY

It turned out that this man had never had a diving

medical. He was on anti-hypertensive treatment. He also had multiple medical problems with irritable bowel and back problems. In 1997 he had a motor vehicle accident where he had blunt chest trauma to his left upper chest, which is where the adhesions were found, but the chest X-ray done straight after the accident was negative.

Similarities between the two cases

There are striking similarities between the two cases, both were novice divers but reported as being very experienced. Both were obese. Both had multiple medical problems. Both were sudden deaths on the surface of the sea from gas embolism. Both were initially thought to be a myocardial event but there was no cerebral vascular event or coronary artery disease found in either one. Both had bitten their tongues which to me indicates that both had had a convulsion. Both had pleural adhesions. They were both diving in a team, they probably had controlled ascents. Their dive medicals were probably inadequate and none.

Both were alone on the surface but I do not think that a buddy would have been any use at any stage during the rescue. Except perhaps they would have been able to tell us what really happened.

They were both recorded by the pathologist as being drowning deaths and on the second one the pathologist, even after we found air in the heart, said "I'll still put this down as a drowning death for insurance purposes". In this man I think his convulsion occurred when he went to inflate his buoyancy jacket. He was floating when he started to convulse and that is why his jacket over-inflated.

Conclusions

These two divers died from drowning, but the cause of the drowning was air embolism, causing unconsciousness, after barotrauma in lungs tethered by pleural adhesions.

It is very important that an experienced diving doctor, with good reasoning abilities attends, every post-mortem after a diving death to integrate such oddities as bitten tongues into the pathologist's diagnosis.

Dr C J Acott FANZCA, DipDHM, is the Co-ordinator of the Diving Incident Monitoring Study (DIMS) and a consultant for the Diver Emergency Service. He is Director of Diving Medicine, Royal Adelaide Hospital Hyperbaric Medicine Unit, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000, Australia. Phone +61-8-8222-5116. Fax +61-8-8232-4207.
E-mail <cacott@optusnet.com.au>.

EFFECT OF HYPERBARIC OXYGEN THERAPY ON LUNG VOLUMES IN NORMAL SUBJECTS AND HEAVY SMOKERS.

Paul Thomas, Clifford Ng, Barbara Trytko
and Michael Bennett

Key Words
Hyperbaric oxygen, physiology, research, lung volume, airway obstruction

Abstract

Decompression from hyperbaric conditions may be associated with air-trapping if the alveoli are unable to empty rapidly enough as ambient pressure decreases. In particular, such air-trapping may occur with the airway obstruction seen in emphysema and asthma. To determine whether this is clinically detectable, subjects with a heavy smoking history and airway obstruction were studied in a hyperbaric chamber.

Fifteen subjects underwent lung volume estimation by body plethysmography at sea level and again immediately after hyperbaric oxygen therapy at 240 kPa for 90 mins. Results were compared with those of seven non-smokers undergoing the same treatment, and five non-smoking staff breathing air.

There were no significant changes in lung volumes in any of the study groups in terms of their lung volumes. The smokers had more airway obstruction than the other groups, but this was mild despite a heavy smoking history. Mild airway obstruction does not appear to be associated with significant gas-trapping after hyperbaric therapy.

Introduction

Patients receive hyperbaric oxygen therapy (HBO_2T) for a variety of medical indications, including osteoradionecrosis, necrotising infections and chronic ulcers secondary to diabetes and vascular insufficiency. An increasing number are elderly and have vascular insufficiency, or have a history of head and neck squamous cell carcinoma secondary to smoking. These smokers or ex-smokers may benefit from HBO_2T, but there are theoretical reasons why acute changes in lung volumes might occur in some subjects with airway dysfunction. Such dysfunction has not been documented, nor have adverse events been reported. It remains possible, however, that airway obstruction in smokers or asthmatic subjects might cause trapping of gas within the lungs as alveolar gas expands on decompression.

It was hypothesised that those with airway obstruction secondary to smoking would have a tendency