Case report

Pulmonary barotrauma and arterial gas embolism from free-ascent training: case report

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Key words
Pulmonary barotrauma, cerebral arterial gas embolism, decompression illness, ascent, treatment, case reports

Abstract

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A 29-year-old military dive trainee suffered a pulmonary barotrauma and arterial gas embolism during free-ascent training. The diver presented acutely with a pneumothorax, subcutaneous emphysema, and evidence of cerebral and spinal involvement. Clinical reports of cases with this particular combination of conditions are rare. Some factors affecting incidence, pathology and outcome are discussed.

Introduction

In the military and civilian environments, ascent training is conducted in order to provide a means by which a diver or submariner can reach the surface in an emergency when alternative breathing equipment is not available. In addition, some elements of the military conduct free ascents during clandestine operations.

This incident involved a military diver who was undergoing free-ascent training at the Submarine Escape Training Facility (SETF) at *HMAS Stirling*. SETF is a six-floor building and contains a 20-metre fresh-water (mfw) tower built for the purpose of providing ascent training to submariners. The water column is well-lit and warm (approximately 34^{o}C). The tower is fitted with recompression chambers (RCCs) at the base and surface of the water column. The RCCs and medical team are available at immediate notice during operations in the tower.

Case report

A 29-year-old, male member of the Australian Defence Force (ADF), who was a qualified but inexperienced self-contained underwater breathing apparatus (scuba) air diver, was undergoing free-ascent training at SETF. Diver X had completed a Navy scuba air diving course 10 years previously but had not dived extensively since. He considered himself to be inexperienced. He had a diving medical seven months prior to this training and met the ADF diving medical standards. In particular, inspiratory and expiratory chest X-ray (CXR) and pulmonary function tests (PFTs) were all within normal limits. During the assessment at this presentation the patient reported a previously undeclared history that he had suffered asthma and pneumonia as a pre-school child. He could not recall any requirement for treatment. He suffers some seasonal hay fever but is free of other illness or injury. He was a smoker between the ages of 14 and 20 years.

Diver X had performed a shallow scuba air dive within seven hours of the incident (38 minutes' duration at four metres' depth).

Free-ascent training was conducted wearing wetsuits and each diver was paired with a supervisor (Figure 1). The supervisor wore an open-circuit scuba air set from which both he and the diver breathed at depth. Ascent was conducted with the divers holding each other, ascending on a jackstay with the trainee's regulator out of his mouth but available. The diver was required to exhale continuously, maintain a posture that extends his airway and swim to the surface at a rate not exceeding 3 sec.m^{-1}.

Within minutes of successfully completing an ascent from 9 mfw and without exiting the water, a second ascent was conducted from 18 mfw. During the 18 mfw ascent Diver X was observed by the accompanying supervisor to be ascending too rapidly and was slowed down. He later described (and was observed from the surface by a diving supervisor) gulping during the ascent at approximately 5 mfw depth.

On reaching the surface, Diver X exited the water via a ladder and was immediately observed to be unable to declare himself well (Figure 2). Within seconds he collapsed and complained of difficulty breathing, generalised tingling and right-sided pleuritic chest pain. He remained conscious with a Glasgow coma scale score of 14–15, but became increasingly agitated and disorientated over several minutes. Initial assessment revealed a right-sided pneumothorax and rapidly progressive neurological

Figure 1
An unaccompanied supervisor demonstrating a free ascent from 18 metres' depth at the Submarine Escape Training Facility at *HMAS Stirling*

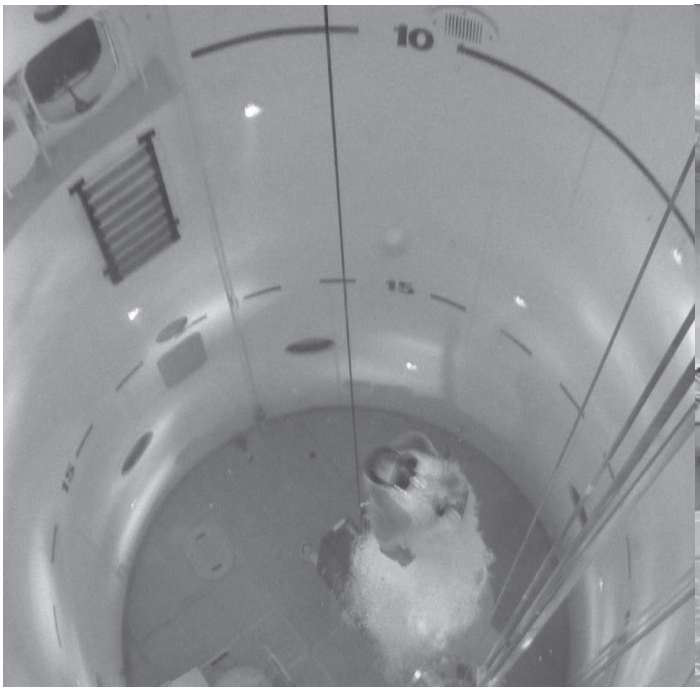


Figure 2
The exit ladder from the water tower with RCC at immediate notice. An instructor is providing a demonstration during Submarine Escape Training.

deficits in both lower limbs including hypertonia, spasms, weakness (graded 1–3/5 in both legs) and incoordination. A presumptive diagnosis of pulmonary barotrauma (PBt) and cerebral arterial gas embolism (CAGE) with right-sided pneumothorax was made.

He was recompressed to 283 kPa within five minutes of collapse, and a Royal Navy treatment table 62 (US Navy 6) was commenced. On reaching 283 kPa and commencing 100% oxygen by mask, his dyspnoea improved and he became well-orientated but continued to complain of significant right-sided pleuritic chest pain and bilateral leg weakness, worse on the right side. Intravenous (IV) fluids were administered and he was aggressively hydrated. He remained haemodynamically normal throughout the treatment.

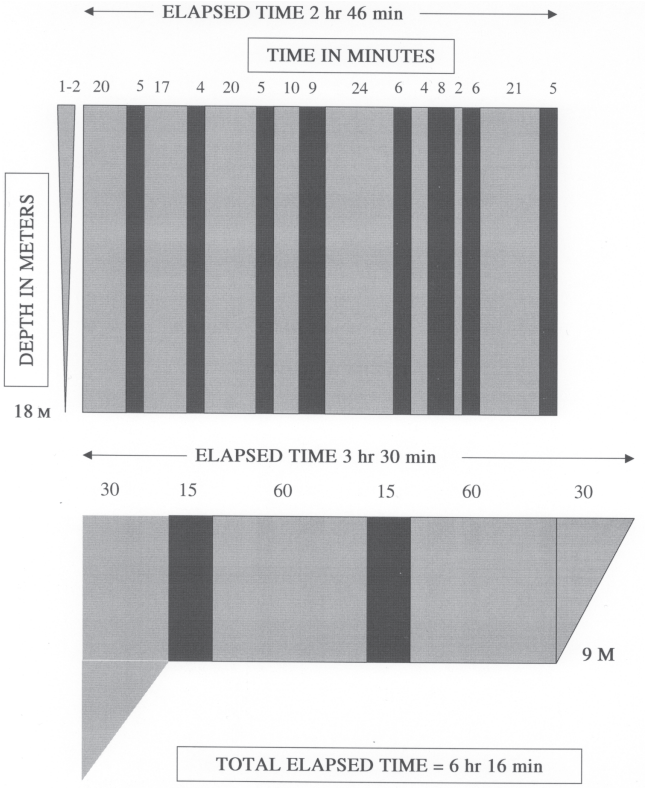
At 30 minutes into the treatment table, examination by the medical officer revealed persistent right-sided pneumothorax, subcutaneous emphysema over the right anterior chest wall from clavicle to nipple, and neurological deficits limited to the patient's lower limbs. Neurological examination revealed a hypertonic right leg with weakness graded at 1–2/5 in his right leg and 4–5/5 in his left leg. Sensory loss was patchy and limited to the lateral aspects of the right thigh and right leg. Knee jerks were hyper-reactive on both sides with absent ankle jerks and equivocal plantar reflexes. An intercostal catheter with underwater seal drainage was inserted to relieve the pneumothorax.

Delivery of oxygen during the recompression was both interrupted and delayed due to a combination of a built-in breathing system failure during the first two oxygen-breathing periods, and the diver's nausea, vomiting and anxiety during insertion of the chest tube. The treatment profile is detailed at Figure 3. It is estimated that at least 81 minutes of effective 100% oxygen was delivered at 283 kPa.

Prior to leaving 283 kPa, power, sensation and coordination had returned to normal and remained so at 202 kPa and on completion of the treatment table. He received no further recompression treatment on the basis that the risks of complications with a chest tube *in situ* were considered to outweigh any potential clinical benefit given his full neurological recovery on clinical examination. CXR the following morning confirmed small residual pneumothorax and subcutaneous emphysema that persisted for several days clinically. The intercostal catheter was removed uneventfully 48 hours after insertion.

Post-injury management included respiratory assessment with CXR, PFTs and high-resolution computerised tomography (CT) of the chest, all of which were repeated at two months post injury. The initial CT showed some residual surgical emphysema but was otherwise normal. PFTs performed at five days post incident and then at two months demonstrated progressive improvement in lung volumes, ventilatory capacity and transfer factor consistent with

Figure 3
Treatment (modified RN 62) for Diver X. Problems with oxygen supply and vomiting interrupted HBOT (grey: oxygen; black: air)



resolution of the small residual pneumothorax, and excluded any on-going respiratory disease. CT was normal at two months. Review by a respiratory physician concluded that he had no respiratory disease or structural abnormalities. Within one week of the accident the diver was back running and has since been returned to full duties but with permanent exclusion from diving.

Discussion

PBt of ascent and CAGE are widely recognised potential complications of compressed-air diving and free-ascent training. The risks of their occurring during submarine escape training and other forms of ascent training have resulted in the discontinuation and/or significant modification of these activities.[1] Although a rare event, PBt with arterial gas embolism (AGE) is one of the more frequent causes of death in scuba diving and arguably may be the leading cause of death in the recreational diving community.[1] Mortality rates in the range of five to ten per cent have been reported even with the institution of appropriate treatment.[1,2]

The two main contributing factors in the incidence of PBt are considered to be pulmonary pathology (e.g., asthma, adhesions and bullous disease) and inadequate technique resulting in air trapping during ascent. PBt and secondary AGE occur more frequently in novice or inexperienced divers.[3] However, most cases of PBt reported in the literature describe the use of apparently appropriate exhalation techniques and an absence of detectable lung pathology on post-injury medical screening.[1,4]

Clinical evidence of pulmonary damage in cases of AGE is rare (3%). Sequelae of PBt include pulmonary tissue damage, pneumothorax, mediastinal and subcutaneous emphysema, and AGE. These may occur singly or in combination. The simultaneous occurrence of these conditions is reported as low.[5] PBt is the result of pulmonary tissue being damaged by a transmural pressure change that causes over-stretching of the lung tissue.[6] Pressure differentials of as little as 70 mmHg across lung tissue can cause tearing.[7] Benton et al report a case of PBt and CAGE from a depth of 1m during helicopter escape training.[8] Others have reported PBt and CAGE from 5 m and less.[9]

Pneumothorax associated with PBt occurs due to air entering the pleural cavity via either rupture of the visceral pleura or by air tracking through the mediastinum and via the parietal pleura. Subcutaneous emphysema results from gas tracking along tissue planes surrounding the airways and blood vessels, into the hilar regions and then into the mediastinum and neck. Subcutaneous emphysema is invariably associated with the existence of a pneumomediastinum.[1]

The neurological signs in Diver X were unusual in that they could represent a combination of cerebral and spinal pathology or a cerebral or mid-brain lesion alone. The acute presentation and rapid deterioration precluded any more than a primary survey and brief neurological assessment prior to recompression of this patient.

Diver X completed a shallow no-decompression dive within seven hours of this ascent that was well within the guidelines of the Royal Australian Navy dive tables. The 'gas burden' from this dive profile, in addition to the surface interval, is small. It is generally reported that the 'submarine escape training presentation' type of PBt and AGE (amongst which this would be included given the lack of nitrogen tissue load and access to immediate recompression) does not produce a combination of decompression sickness (DCS) and AGE pathology.[1] In addition, spinal cord DCS is not thought to be principally embolic, since AGE is more likely to affect the more vascular grey matter of the cord rather than the white, and the effect of venous gas emboli could not account for the rapid presentation in cases like this.[10]

Neuman describes a syndrome of 'biphasic DCS' that occurs in the setting of an AGE and a trivial gas burden and may explain the presentation in this case.[1] It is suggested that AGE can precipitate DCS in divers who are well within the US Navy no-stop limits and who would not otherwise be expected to suffer DCS.[1] These cases are reported as severe and refractory to treatment.

MEDICAL SCREENING

PFTs are used in divers to detect pulmonary pathology and thus minimise exposure to risks such as PBt and AGE. An association between pre-morbid low forced vital capacity (FVC) and PBt has been reported in submarine escape trainees.[11]

Consistent with Australian/New Zealand Standard 2299.1 and other diver medical screening recommendations, the ADF medical screening for diving requires spirometry to be performed.[12,13] This is despite a lack of evidence that the incidence of PBt among divers has been reduced through this screening.[14] Forced expiratory volume in one second (FEV_1) and FVC values within the predicted normal range do not exclude an individual from developing PBt.[13] There is some limited evidence that mid-expiratory flow rates, derived from a single-breath flow-volume loop, may be more predictive of PBt.[15] However, numbers in that study were small and definitive recommendations cannot be made without further research.[16] In Diver X, simple spirometry, not a flow-volume loop, was conducted prior to diving. His post-morbid results, which include mid-expiratory flow rates, were all within normal limits.

Recommendations regarding diving with a history of asthma remain controversial due to the lack of controlled clinical data available. Current ADF policy is to exclude from diving members with evidence of demonstrable bronchial hyper-responsiveness. Although many experts consider that an absence of symptoms in the last five years is a reliable indicator of the absence of significant bronchial hyper-responsiveness, it has also been demonstrated that a proportion of individuals with demonstrable bronchial hyper-responsiveness do not have a clinical diagnosis of asthma.[17] ADF diving candidates who declare a childhood history of asthma but have been asymptomatic since are generally referred for provocation testing.

TREATMENT

Immediate treatment of PBt occurring with AGE includes resuscitation, hyperbaric oxygen therapy (HBOT), intravenous fluids and consideration of adjunctive treatments such as lignocaine. It is widely accepted that the early application of HBOT plays an important role in the management and treatment of AGE and that delays to recompression significantly increase morbidity and mortality.[18] Delays in recompression exceeding four hours reduce the chances of complete resolution by 50 per cent.[19] Gorman concluded that if the delay to recompression following AGE is less than five minutes then the morbidity in survivors is almost zero although five percent may still die.[2]

Similar injuries occurring in the open-water environment are reported as having poorer outcomes and this is largely explained by the increased complications of near drowning, delays in recompression and resuscitation, and additional nitrogen gas burden. The excellent outcome in this case can be attributed to the extremely controlled environment in which the training was being conducted and the immediate access to recompression facilities.

Conclusions

This case is a rare presentation of PBt and AGE. The following factors could be considered as potential contributors: gulping during the ascent (poor technique, relative inexperience and anxiety); microstructural pulmonary pathology; and possible bronchial hyper-responsiveness (childhood history of asthma). Mitigation of PBt/AGE in this context is addressed through the conduct of best practice and evidence-based medical screening, the prompt availability of trained medical support and recompression, and delivery of high-quality diver training. It cannot be concluded that additional medical screening would have helped to prevent this case and it is not possible to determine the cause precisely. The excellent clinical outcome is most likely attributable to immediate access to recompression facilities and medical care, and to favourable physiological characteristics of the patient.

Acknowledgments

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Divers Alert Network Project Dive Exploration

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Liveaboard diving, at just one case of decompression illness (DCI) per 10,000 dives, is providing the lowest incidence of DCI cases, according to a continuing research programme being carried out by Divers Alert Network (DAN). For whatever reason, the highest ratio has been for divers visiting Britain's Scapa Flow, at 30 DCI cases per 10,000 dives.

DAN, the international diving medical research and advisory body, is seeking the help of sport divers all over the world to research the causes of DCI. Rather than using scientific theory to work towards a diving decompression model, its Project Dive Exploration (PDE) aims simply to record which dive profiles worked without problem for divers and which did not. When enough data has been gathered, DAN hopes to produce tables and computer models which will give an idea of the DCI risks for different dive profiles, based on experience. Such a model would probably be regarded as complementing rather than replacing existing decompression programmes.

DAN developed its PDE data collection methodology and software from 1995–97, and began collecting data in 1999. So far, some 105,000 dives, whether safe or involving injury, have been logged and analysed. It is from these preliminary figures that the Scapa and liveaboard data are derived. DAN plans to obtain and work through more than a million responses before it begins to establish any sort of definitive model for divers to use. The scale of the programme makes PDE the "*most extensive study of recreational diving ever conducted.*" For each dive they do, participating divers fill out a downloadable form, upload their dive computer's dive record (or otherwise record the profile if they used a table), and provide details of their physiology and health. A 48-hour post-dive report is included. Here the diver lists any medical signs and symptoms, or recompression treatment received.

If a chamber is involved, DAN will contact it for medical details, and find out whether flying after diving was involved. It has been estimated to take 30–60 minutes for a diver to send to DAN details relating to, say, 20 dives on a liveaboard trip. DAN is conducting PDE "*wherever diving occurs throughout the world.*" Divers can obtain relevant information on how to participate from dive centres, shops or aboard charter boats, or visit DAN's website, www.diversalertnetwork.org, or the South-East Asia Pacific offices at www.danseap.org. SPUMS members are encouraged to join the PDE programme themselves as well as encouraging divers they see in the course of their work to do the same.