Case reports

Arterial gas embolism breathing compressed air in 1.2 metres of water

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Abstract

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Arterial gas embolism (AGE) may result when diving while breathing compressed gas and ascending rapidly or with a closed glottis. Pulmonary over-pressurization can result in lung stretch injury with entry of bubbles into the pulmonary venous circulation and subsequently the systemic arterial circulation. We present the case of an individual who suffered AGE while breathing compressed air at 1.2 metres' fresh water (mfw) in a swimming pool and discuss the factors determining the depth at which this form of injury may occur. This case serves to underscore the fact that risk of AGE exists at shallow depths.

Introduction

A recognized complication of diving while breathing compressed gas is pulmonary barotrauma.[1] Pulmonary over-pressurization and barotrauma takes many forms, ranging from pulmonary interstitial, mediastinal, or subcutaneous emphysema to pneumothorax and arterial gas embolism.[2] Arterial gas embolism (AGE) is believed to result from disruption of the pulmonary venous circulation and entry of gas into the vasculature.[1] Divers are often unaware of the depth at which such problems may occur, believing that limiting excursions to a relatively shallow depth removes all risk. The exact depth required for AGE to occur remains undefined. The purpose of this report is to present the case of an individual who developed AGE following a brief surface-supplied dive at 1.2 metres' fresh water (mfw) in the shallow area of a swimming pool. Additionally, we provide a discussion of the potential factors that contribute to the pathophysiology of pulmonary barotrauma (e.g., AGE) at shallow depths.

Case report

The patient was a 25-year old healthy, fit military aviator referred for treatment of AGE. On the day of his injury, he participated in a training exercise in a swimming pool. While wearing his flight suit, he first sat on the floor in the shallow area of the pool (120 cm water depth) and breathed compressed air for about one minute. The breathing gas was supplied by a compressor located on the pool deck which was connected by a hose to a regulator and then three additional feet of hose to his mouthpiece. When sitting on the bottom, the top of his head was just below the water's surface. This exercise was accomplished uneventfully. He then exited the pool and re-entered it, this time hanging upside down by his knees from the pool's edge, again breathing from his mouthpiece. He was head-down in this fashion for a total of approximately three minutes. To demonstrate how a regulator works, he was asked to move it vertically in the water, sensing less pressure when moved toward the surface and more pressure when it was moved deeper. He recalled moving the regulator to the bottom of the pool, disengaging his knees from the pool's edge and standing up. Total time in the water was estimated at four minutes. He felt entirely well during exit from the pool, then experienced the onset of vertigo while stowing his gear. This was associated with left temporal headache which progressed rapidly to bitemporal pain. He estimated onset of symptoms within five to ten minutes after exiting the pool. He was transported immediately to the base medical clinic where evaluation demonstrated 'unsteady Romberg test and mild difficulty with finger-to-nose testing.' High flow oxygen administration, intravenous fluids and 25-degree head-down positioning were initiated by the on-site medical staff, and the patient transported by ground to the emergency room of a hospital with a multiplace hyperbaric facility.

Upon arrival, history and physical examination were notable for complaints of headache over the top of his head self-rated at 7–8/10 and pressure in the left ear without evidence of otic barotrauma om examination, as well as 'positive Romberg testing with falling to the right'. With a working diagnosis

of AGE, he was treated on a US Navy Treatment Table 6A with full extensions. Upon completion, headache was rated at 1/10, sensation of ear pressure improved and dizziness described as only slight. Neurological examination was normal. The patient had a very slight sensation of dizziness for 48 hours, then experienced resolution and felt entirely well.

One day prior to the event, he had participated in an aircraft decompression exercise. He began breathing 100% oxygen by face mask on the runway, flew to 15,000 feet altitude decompressed his aircraft to ambient, re-pressurized the aircraft to 8,000 feet, and ascended to 40,000 feet. He remained on oxygen throughout the flight and felt well afterward. The following morning, he awoke with the sensation of pressure in both ears, cleared them with a Valsalva manoeuvre and felt normal.

Subsequent evaluation included a normal chest radiograph and pulmonary function testing. The latter included plethysmographic measurement of lung volumes that demonstrated total lung capacity of 8.94 L (119% predicted), vital capacity 7.10 L (120% predicted), residual volume 1.85 L (105% predicted) and normal airways resistance. Other measurements included the distance from the patient's knees to his mouth (81 cm), pool deck to the surface of the water (20 cm) and pool deck to the bottom of the pool (140 cm).

Discussion

This individual is believed to have suffered cerebral AGE while surface-supplied diving at a depth of 1.2 mfw. The diagnosis is supported by his history, temporal onset of symptoms after emerging from the water, physical examination findings and response to recompression therapy. Other diagnoses to consider in the diver with acute neurological symptoms and signs can be effectively excluded on clinical grounds in this case. This was not decompression sickness due to the brief duration and shallow nature of the diving exposure. Inner ear barotrauma could cause dizziness and imbalance but would not have been expected to respond to recompression therapy. A transient ischemic attack due to a thromboembolic event would be extremely unlikely in a healthy, fit military aviator of this age and, again, would not temporally respond to hyperbaric oxygen treatment.

His case is remarkably similar to the one reported by Benton in 1996, also a military aviator who developed AGE while undergoing training in a swimming pool.[3] In that case, the diver was limited in depth to one meter and was not inverted. He suffered multiple neurological symptoms immediately upon exiting he water, including upper extremity paraesthesias, subjective diplopia, and objective memory loss upon testing. He required repetitive hyperbaric treatment but eventually resolved all symptoms and signs. Subsequent pulmonary function testing and thoracic imaging were normal and he was cleared to return to flying.

Whether lung rupture occurs during diving with compressed gas breathing depends upon several factors, including lung compliance, transpulmonary pressure, and lung volume.[4] Some small degree of pulmonary over-pressurization can be accommodated by lung expansion, diaphragmatic inversion, and compression of the heart and intrathoracic veins.[5] Lung rupture occurs when pulmonary parenchyma is stretched beyond its limits and is subsequently torn by over-pressurization.

Schaefer and co-workers performed experiments involving decompression of dogs from 100–200 feet' seawater over 60–90 seconds with the trachea closed.[6] Animals developed pulmonary interstitial emphysema and AGE when the intratracheal pressure reached a critical value of approximately 10.7 kPa (80 mmHg) or a transpulmonic (intratracheal minus intrapleural) pressure of 8.0–9.3 kPa (60–70 mmHg). In the classic 1961 article of Malhotra and Wright, two fresh human cadavers aged 47 and 64 years were demonstrated to develop pulmonary barotrauma when the lungs were pressurized to intratracheal pressures of 9.7 and 10.7 kPa (73 and 80 mmHg) while the thorax was unbound.[2]

What is required to achieve these pressures while diving? If the intra-alveolar pressure were 0 mmHg and the respiratory system compliance 0 ml·mmHg^{-1} at total lung capacity (TLC), this pressure could be achieved at sea level by adding one-tenth of an atmosphere absolute (10.1 kPa [76 mmHg]) to the system, an equivalent depth underwater of one metre. As such, this is the depth commonly proposed as the lower limit at which AGE can occur.[1,4,7]

However, during breath holding with closed glottis at TLC, intra-alveolar pressure is already elevated due to elastic recoil of the lungs and chest wall. When measured by Rahn in 14 subjects, the average intra-airway pressure was 2.8 kPa (21 mmHg) above ambient pressure at TLD.[8] Colebatch made the same measurement in a group of ten healthy young adults and reported an average of approximately 4.3 kPa (32 mmHg).[9] If pressure were the only determinant of pulmonary rupture, these data suggest it may be possible for AGE to occur at a minimal depth; even less than one metre in the case of a diver who ascends with full inspiration and closed glottis.

The volume of gas necessary to consistently cause demonstrable lung stretch injury has been assessed in a group of breath-hold divers. Chung and co-workers found evidence of pulmonary barotrauma (mediastinal emphysema on computerized tomography) in each of five divers proficient at adding gas volume to their vital capacity (VC) through gastroesophageal insufflation (GI).[10] Prior to imaging they used GI to add an average of 1,400 ml (26%) to their VC. As the VC represents approximately 80% of TLC, it is apparent that a 20 % increase in TLC (0.8 x 26%) may be sufficient to cause lung stretch injury, suggesting a maximal depth limit of two meters for predictable lung injury to occur.

These calculations assume that the lung is a homogeneous structure with uniform compliance throughout. However, studies of regional ventilation show significant heterogeneity which most likely indicates variability in lung compliance and airway resistance.[11] Such heterogeneity could be caused by prior local infection or exposure to external irritants or toxins. There are instances of similar changes in transpulmonary pressure causing AGE, often in individuals with pre-existing lung pathology and changing altitude.[12]

In this case, the diver was breathing from the regulator while inverted and head-down with his head near the bottom of the pool. Although he recalls moving the regulator to the pool floor before surfacing, it is not known whether he inspired to TLC prior to disengaging his knees from the pool edge and standing up. If he did, it is possible that immediately before surfacing his lungs were 'pre-stretched' due to increased airway pressure resulting from elastic recoil, as well as a positive static lung load resulting from his regulator being situated at a deeper depth than his lungs.

In summary, the depth at which a diver breathing compressed gas is at risk for pulmonary barotrauma is somewhat individualized and not simply based upon intrapulmonary pressure. It also depends upon the degree of inspiration prior to breath-hold ascent, as well as heterogeneity of the lung and how comparable a healthy diver's lung tissues are to those of canine models and middle-aged cadavers. The minimum depth at which there is the possibility of pulmonary barotrauma resulting in AGE may even be less than the one metre quoted under certain circumstances.

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