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AIR EMBOLISM - A DISCUSSION BASED ON TWO DIVING ACCIDENTS

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Arterial gas embolism (AGE) occurs in divers, submarine escape trainees and in patients having open-heart surgery or even arteriography. It can occur in people who have suffered venous air embolism if there is a patent foramen ovale or a vascular anomaly in the lungs which by-passes the lung capillaries. Margery Allingham had one of the characters in "The Mind Readers" die from arterial gas embolism when a "sparklet" operated cork remover pierced his chest and heart.

CASE HISTORIES

Case 1

An experienced sports diver in his late 20's finished a decompression dive and ascended normally. He is sure that he was breathing in and out all the way up and came up not faster than 60 feet a minute. On the surface he found swimming difficult as his legs were suddenly weak and difficult to control. He reached the boat and had to be helped in as his arms had become weak. Once on board he collapsed. He could hear people talking but could not see. To the onlookers he was unconscious. He was hyperventilating and having occasional extensor-spasms. He was like this for more than half an hour. Then he woke up and appeared normal except for his left leg. The others in the party, occupied with packing up, let him wander off and drive himself home. A couple of days later he contacted me because he kept on missing his mouth with his teacup and missing the saucer when he put the cup down. Recompression cured him. This case has been reported in more detail in the SPUMS Journal of April-June 1983.[1]

Perhaps the diagnosis of the doctor (another diver) on board the dive boat, that his troubles were due to hyperventilation was right but as hyperventilation follows iatrogenic arterial gas embolism I prefer to explain his problems with an air embolism.

Case 2

A young woman was finishing her diving training course by doing open water dives. The first dive was uneventful. Back in the boat after the second dive she said that she was not well, then collapsed, stopped breathing and went blue. Mouth to mouth resuscitation was started and after about five minutes she started breathing and regained consciousness. About 10 minutes later she went unconscious and stopped breathing. Once again expired air resuscitation was successful. Soon after the boat arrived in port and she was transferred to hospital. There she had no demonstrable neurological abnormalities.

SETT experience has shown that the symptoms of air embolism may come on minutes, sometimes many minutes, after leaving the water. Those on board thought that she had had an air embolism and I agree with them. I believe that her second collapse was a second embolism brought on by sitting up.

People have died ascending from as little as two metres without breathing out and the standard explanation is that pulmonary barotrauma occurs when the lung is over stretched. As a consequence of the lung bursting air can find its way into the interstitial tissues of the lung and of the mediastinum, down below the diaphragm retroperitoneally or intra peritoneally, up into the neck, into the pleural cavity and also into the pulmonary venous system. These latter bubbles then pass through the heart and are pumped out into the aorta and so reach the brain causing arterial gas embolism. It is a reasonable explanation.

We know that human corpses burst their lungs when intrapulmonary pressure, measured in the trachea is about 90 mmHg, which is roughly 1.2 msw.[2] Binding the chest and abdomen allowed the pressures to be approximately doubled before the lung burst. Admittedly Malhotra and Wright only reported 5 experiments, one unbound and four bound cadavers. The unbound body developed a pneumothorax while the others developed pulmonary interstitial emphysema. As they had no circulation none developed air emboli.

The majority of studies of air embolism come from Submarine Escape Training Tanks (SETT). Large numbers of men perform these buoyant ascents every year and a few suffer pulmonary barotrauma and air embolism. Very few have evidence of interstitial emphysema or of pneumothorax. The ascent rate is very rapid. At HMS DOLPHIN the trainee is clipped onto a wire rope running up the centre of the 100' tank to make sure that he does not hit the wall and damage the paintwork, or himself. In spite of the friction of the clip on the rope the survival suited figure is travelling fast enough for those at the top of the tank to see clear between his legs before he drops back into the water. Sports divers do not achieve this speed of ascent. They also wear wetsuits which, in many cases, splint their chests and abdomens. So it might be that SETT trainees and sports divers acquire their air emboli differently.

There are a number of inconsistencies about pulmonary barotrauma and arterial gas embolism. The pages in the 1973 Bennett and Elliot that deal with air embolism suggest that haemoptysis, signifying lung damage, is common.

Macklin reported pneumothorax with massive collapse from experimental over inflation of the lung in cats in 1937.[3] He jammed a catheter into a bronchus and was rewarded with massive air leaks into the lung perivascular spaces, the mediastinum, the retroperitoneal tissues, the neck and the pleural space. What he did NOT report was air in the pulmonary vessels.

Macklin and Macklin published a long paper (77 pages) in 1944 on "malignant interstitial emphysema of the lungs and mediastinum".[4] It was malignant because it led to an anoxic death after steadily increasing dyspnoea and great inspiratory efforts. Their conclusion was the overdistention of alveoli adjacent to blood vessels allowed air to enter the potential space around the blood vessels with each

inspiration and each expiration pushed this air towards the hilum, so stiffening the lungs and making the dyspnoea worse. The process was self perpetuating. Certainly increasing dyspnoea, cyanosis and death was a common ending in the influenza pandemic of 1919 and with pneumonias before antibiotics.

When an anaesthetist forgets to release the expiratory valve during controlled ventilation the usual result is surgical emphysema of the neck or a pneumothorax. Yet both these manifestations of pulmonary barotrauma are much less common in divers and SETT trainees than is AGE, which in some parts of the world is the major pressure-related problem of diving.

In the SPUMS Journal of July to September 1983, Takashi Hattori discussed ten years of diving accidents on the Monterey Peninsula in California.5 Only 20% of his cases of AGE had haemoptysis. Hattori treated more cases of AGE than of decompression sickness between 1971 and 1981. In 1982 Harry Oxer presented 6 pressure related accidents from Western Australia to a SPUMS meeting in Melbourne. Five were AGE and one was a case of high altitude decompression sickness.[6] In Victoria there are approximately equal numbers of cases of AGE and decompression sickness.

It is easy to understand how part of the lung, either partly or totally blocked off from the airways, can be over expanded and rupture, tearing capillaries and so allowing air to enter them if they remain open. It is less easy to imagine the whole lung behaving this way. In fact a friend of mine was making an out of air ascent many years ago. Suddenly his regulator was blown out of his mouth. He does not remember anything more of the ascent till he "woke up" on the surface. He had a bit of a cough for a while but felt well enough to have another dive that afternoon. Certainly he had an over pressure accident. Was his unconsciousness due to a faint caused by intrapulmonary pressure interfering with cardiac filling or was it due to an air embolus? No one knows.

The lack of X-ray changes and of pathological changes in the lungs of clinical air embolism cases treated at the Royal Australian Navy School of Underwater Medicine led Carl Edmonds to refer these people to Professor Colebatch for exotic respiratory function tests. Professor Colebatch found that there was evidence of differences in compliance in different parts of the lung in these people.[7] This is unusual. The testing is time consuming and complicated and quite unsuited as a screening test for divers. His explanation for AGE in some of these people is that at the boundary between the normal and abnormally stiff parts of the lung expansion of the normal lung, being more than that of the stiffer lung, tears some of the alveoli between the two areas. This of itself is of no great significance as the intrapulmonary pressures, though raised, are in the normal ratio.

However when the diver, who was out of air, reaches the surface and gasps in his first breath, the normal lung expands maximally and pulls away from the stiffer lung, which holds open the holes in damaged venules and capillaries. Air enters the damaged area, as it does the rest of the lung, on inspiration. On expiration it cannot leave and either goes up the perivascular spaces towards the hilum or enters the damaged blood vessels. His contention is that it requires a deep inspiration to either damage or overfill the damaged area between the normal and abnormally stiff parts of the lung. This is an interesting and appealing hypothesis and explains the following case.

One of my patients developed a pain in his chest doing a series of forced vital capacity tests on a Vitalograph. So I had his chest X-rayed and his mediastinum was very neatly outlined with air. I assumed that he had burst his lung while blowing, but could not understand how or why. When I discussed the occurrence with Professor Colebatch he thought that the maximal inspirations had done the damage and that the forced expiration had pushed the air towards the hilum, which is at least believable.

It seems more likely to me that a recurrence of symptoms following an air embolus is a new embolus rather than anything else. After all the lesion in the lung cannot have healed in minutes or even hours. A clot has probably formed within minutes but the bleeding would presumably distort the lung architecture and compliance so transferring the original problem to some other part of the lung. It is quite likely that there is interstitial air in or adjacent to the damaged lung.

To me the interesting things about AGE are:

(1) that it is often diagnosed as hyperventilation by doctors inspite of very obvious neurological signs;
(2) that it occurs much more often than the other manifestations of pulmonary barotrauma;
(3) if Professor Colebatch is right about the mechanism of air embolus in people with "normal" lungs why do only some have relapses?

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