



## TRAUMATIC AIR EMBOLISM

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### Summary

**A rare case of traumatic air embolism and a possible hazard to underwater divers using SCUBA (self contained underwater breathing apparatus) method are herein described.**

### Case History

A 27 year old Austrian 'fit enough to pass all the tests for a pilot with the Austrian Air Force' on an underwater tour using SCUBA, accompanied by his trainer, went down on his fourth dive to 30 meters. He had had a good meal a few minutes before diving. The air cylinders, with no spare one, had just been checked. While under water the trainer noticed that his companion was 'shaking' and had removed the mouth piece from his lips. The trainer put his mouth piece on to his companion's lips and quickly ascended. The attending boat picked them up but the Austrian was 'stiff all over', unconscious and had to be placed flat on his back across the boat. Being rowed ashore vomit started coming out of his mouth and he was 'breathing with difficulty and noisily'.

On arrival at the Victoria Hospital, Gozo the patient was breathing stertorously, slightly cyanosed, rigid and shaking all over. He was unconscious, had dilated pupils and his eyeballs were roving from side to side. Signs of vomit were on his left cheek and chest wall. His hands were in the 'main d'accoucheur' position and his feet were in state of exaggerated plantar extension. No signs of external bodily injury were evident and there was no surgical emphysema. His pulse was faint and very rapid, over 120 per minute. An attempt to take the blood pressure was made. Though the clonic movements interfered with correct reading, the systolic was found to be about 80 mm Hg. The patient was reacting

to painful stimulation by a withdrawal reflex and an attempt to flex his forearms showed a lead pipe type of rigidity. Ankle clonus was present and the reflexes at his knee and ankle were brisk; the plantars were normal and there was no neck rigidity.

There was adequate air entry in the lungs but a lot of extra respiratory sounds were also present. The heart sounds were faintly audible and there was an uncommon murmur heard all over the precordium.

### Treatment:

Although the diagnosis was still in doubt a cuffed endotracheal tube was passed with some difficulty and suction applied through another tube to clear all the vomitus. Then oxygen was given through a Y connection, the other end being left open to the air. An open vein was secured and an infusion of 5% dextrose started. In an attempt to relieve the clonic fits 10 mls of calcium gluconate were slowly given and also 100 mls of sodium bicarbonate 8.4%. There was no improvement in the fits by these methods though the breathing improved and the endotracheal tube was removed. Diazepam in 5 mgs doses was given slowly in the i.v. drip and the fits stopped after the third dose (total 15 mgs). Consciousness returned and, through an interpreter, the patient said he had severe burning retrosternal pain. The blood pressure was again checked and was found to be 120/80 mmHg. Ophthalmoscopy at this stage revealed a pale field and though a special search was made for any air bubbles, none was found. It was noticed that whenever the patient was disturbed or moved clonic fits with opisthotonus occurred, causing the patient a lot of muscle pains. The patient was then moved to the X-ray room and a chest view was taken lying down. There was good expansion of the lungs, the heart shadow was in

place but a rather wide mediastinal shadow was noted.

An examination was again carried out when the patient was back in bed. He could move both his upper limbs but only the right leg. The pupils returned to normality and reacted to light but vision was limited to perception of light.

The provisional diagnosis of 'bends' was made and arrangements were made to transfer the patient to the air compression chamber kindly lent by the British Naval authorities at St. Angelo in the Grand Harbour. Within three hours of the accident the patient was in the chamber. The pressure was set to three atmospheres and the patient complained of severe ear ache. He still had occasional clonic fits when disturbed and repeated doses of Diazepam were given intramuscularly, the intravenous drip having come off.

After eight hours in the chamber the patient died.

#### Post Mortem

At post mortem severe cyanosis was noticed in the upper chest and face, petechiae were present and surgical emphysema involved the neck and chest wall. On opening the thoracic cavity air was noticed in the myocardial vessels, the pulmonary arteries and the heart. The arterioles over both lungs were also peppered with air bubbles. On opening the skull multiple air bubbles were to be seen in the thin walled cerebral blood vessels especially the basilar which was empty of blood (though the patient was all the time lying face upwards) and the right middle cerebral artery.

The postmortem results conflicted with the provisional diagnosis of 'bends' and a massive air embolism was proposed as the cause of death.

#### Discussion

How could air embolism have happened while under water? The pressure of air at sea level is 1 Atmosphere (14.7 lbs per 1 sq.inch). Boyle's law states that the temperature being constant the volume of a gas varies inversely as the pressure. At 33 meters depth (3 more than in this case)

the pressure is three atmospheres or 44.1 lbs per 1 sq. inch. The volume of gas in the lungs was 1/3 its normal value. As the depth is decreased the volume of air in the lungs is proportionately increased. If the vocal chords are kept closed and air is not allowed out overinflation of the lungs occurs. If a weak spot exists in the lung parenchyma a rupture may occur with the opening up of blood vessels and aspiration of gas in the pulmonary vessels and dissemination of air emboli in the peripheral circulation. If the intra pulmonary pressure in the overdistended lungs exceeds ambient hydrostatic pressure by 60 mm Hg air embolism may occur. The underlying mechanism is a pressure gradient from air in the alveoli to perivascular sheaths or underlying septa. Air may rupture blood vessels or dissect along sheaths and septae and through subpleural tears into the mediastinum with such complications as subcutaneous or surgical emphysema.

The most serious complication leading to loss of consciousness and focal seizure with paralysis of one or more limbs implies the entry of air in the cerebral circulation. The shifting nature of this paralysis follows the movement of air bubbles causing patches of ischaemia which if prolonged can permanently damage the brain even though the air bubble moves to smaller and smaller blood vessels.

The volume of air that must have entered the peripheral circulation of the case described above must have been considerable. The low blood pressure and the chest pain suggest severe myocardial ischaemia which improved spontaneously as the air bubbles moved to other smaller vessels.

#### Prevention

This accident illustrates the risk untrained and unprepared divers run when faced with an emergency while deep under water. The trainer accompanying this man suffered from no ill-effects (excluding any diagnosis of 'bends') despite the rapid ascent. The importance of not diving after a hearty meal is made evident by the probability that the vomiting caused this diver to pull off the mouth piece while 30 meters

underwater. Three Atms pressure is more than enough to force the contents of a full stomach up the oesophagus.

Taking an untrained person down to thirty meters on his fourth dive is in my opinion too risky as all 'crash courses' are! To allow air to escape whilst underwater implies a lot of training to overcome the fear that sea water will flood the air passages. Training will help to give confidence and to handle emergencies better. Cardio-pulmonary fitness is most important. Periodic chest X-rays are called for and an electro-cardiogram while performing exercise, thus revealing any ST segment depression, is needed before the start of a season. The maintenance of clear sinuses and open Eustachian tubes calls for periodic visits to ear, nose and throat specialists, if barotrauma is to be avoided.

Though the patient discussed was once

found fit to be an aeroplane pilot this does not exclude the presence of any pulmonary inflammatory lesion that could have caused a weakening of the tracheo-bronchial tree.

### Treatment

Recompression therapy with oxygen inhalation usually brings rapid recovery if the interval between the onset of the accident and the pressure treatment is short. Placing a patient in the left lateral position and head tilted down ensures this displacement of air to the apex of the right ventricle and allows blood to occupy the outflow tract of the ventricle. Methods of directly aspirating air from the heart are described in books but are impracticable. Recompression brings about a decrease in volume of the air bubbles and allows their easy movement and resorption.