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CASE REPORT

AIR EMBOLISM IN A SPORTS DIVER

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INTRODUCTION

We describe a case of air embolism in a sports diver.

The recent increase in numbers of people partaking in diving as a sport, necessitates more staff at the local level becoming capable of at least the initial emergency treatment of diving emergencies. The other factor which needs attention is the availability of decompression chambers on or near the site of the sport to cope with such emergencies. These chambers are surprisingly few and thinly scattered around the country.

In this case, the initial difficulties in diagnosing the extent of the condition caused some delay in calling the Accident Flying Squad to attend and in instituting the correct treatment; fortunately without serious consequences. There was a decompression chamber on site at the scene and without it, it was felt unlikely that the patient would have survived.

CASE REPORT

A moderately experienced eighteen year old female sports diver was diving in a flooded gravel pit (fresh water) and had been at a depth of twenty-two metres (72 ft.) for approximately seven minutes when her mouthpiece ceased to function correctly. She made an emergency rapid ascent from this depth during which she was thought to have inhaled water.

At the surface she was found to be apnoeic, cyanosed and unconscious. However, she responded quickly to

Fig. 1: The site of the dive.

mouth to mouth resuscitation and was taken ashore in the head down and legs elevated position. At this time she had no symptoms and it was arranged for her travel by car to hospital for a "check-up". She then sat up and suddenly developed pain in the right shoulder with associated weakness of movement, visual impairment and reduction in sensation, paraesthesiae and weakness of both legs. She was immediately transferred to the decompression chamber available at the site and at this point, the Leicester Royal Infirmary Accident Flying Squad was called out.

Fig. 2: The therapeutic decompression chamber. Note the very limited space.

None of the members of the Flying Squad had any experience of diving personally. A member of the team could not enter the chamber without risking problems, in particular nitrogen narcosis. There was also lack of space in the chamber and uncertainty as to the length of time the patient ought to stay in. Subsequent management was, therefore, based on the history, a very distant view through a porthole of the patient and an intercom connection with the experienced diver in the chamber with the patient. The medical staff at HMS Vernon were contacted for their advice in the initial management. Later they offered direct help by sending one of their team members to enter the chamber and assess the patient directly. The treatment regime was set up with intravenous Dexamethasone 12 mg, followed by 8 mg six hourly and immediate recompression to an equivalent depth of 50 metres (165 ft. sea water) on air for thirty minutes. Thereafter the patient was slowly decompressed following a fairly standard table.

With this management, immediate improvement and gradual full recovery occurred. The patient left the chamber after 37½ hours and then transferred to the Leicester Royal Infirmary where a CAT scan, chest X-ray and thorough neurological examination was carried out. These did not reveal any abnormality. She was discharged home forty-eight hours after her admission to the hospital with no sign of any residual neurological impairment.

DISCUSSION

Air embolism is one of the complications of the "Barotrauma of Ascent".

As the ambient pressure decreases during ascent, the lungs will tend to expand in accordance with Boyle's Law (see Fig. 1). Hence an ascent from twenty-two metres to the surface would give rise to a possible 2.1

fold increase in gas volume within the lungs. Usually a diver should compensate for this either by continuous exhalation during the ascent or by using the mouthpiece and air supply (which if functioning correctly automatically regulates inspired air pressure to the ambient pressure).

Fig. 3: Boyle's Law — the effect of pressure increase on gas volume.

If insufficient gas is expelled from the lungs, there comes a point when the gas volume and/or pressure in the alveoli will increase to such an extent as to damage or rupture the alveolar sac(s). This extra alveolar air may give rise to several conditions although the patient may be completely asymptomatic.

1. The air may remain in the interstitial tissues of the lung.
2. Dissection may occur along vessels to the mediastinum and on to subcutaneous tissue — often giving only minor discomfort.
3. Pneumothorax may occur — where this occurs, treatment should be according to normal practice, but a Heimlich valve is preferable to an underwater seal drain which functions poorly in the chamber.
4. Air may enter the pulmonary vessels due to sudden large negative intra-thoracic pressure, and then become arterial emboli — producing widespread effects. This is the air embolism, which presumably happened in the patient we have described above when she sat up.

The air embolism causes its effects by mechanical blockage, the vessels most commonly affected are the cerebral arteries. The bubble of air is stabilised by surface tension, and because of this, diffusion and eventual collapse of the bubble is slow. Clinical signs

obviously depend on the size of the bubble and the blood vessel concerned, and the site of the cerebral circulatory occlusion.

Common symptoms are focal paraesthesiae and visual upset. The typical history of such a patient is of an emergency ascent, a sudden gasp for breath on reaching the surface and then unconsciousness. It is the gasp for air on reaching the surface which allows the free air in the lung tissue to enter the circulation by lowering the intra-thoracic pressure. As this is associated with a marked increase in venous return and cardiac output, the air will easily cross damaged capillary walls. Failure to recognise this and delay in treatment may result in the death of the diver.

MANAGEMENT

The following regime is usually recommended for this type of case:—

1. Immediate recompression to 50 m (165 ft. sea water) (= 6 Atmospheres pressure) breathing air for 30 minutes. This is believed to decrease the size of the

bubbles and allow them to move further along the arterial tree. It also facilitates absorption of the bubbles.

2. Intravenous Dexamethasone — in large doses, to prevent cerebral oedema, which may be delayed up to 12 hours (12 mg stat followed by 8 mg 6 hourly).
3. The pressure is then reduced gradually in stages to 18 m (60 ft. sea water) — allowing oxygen to be breathed without great risk of its toxicity.
4. The decompression procedure continues according to the standard programmes.
5. When appropriate the patient should have a chest X-ray to exclude mediastinal emphysema and/or a pneumothorax, a CAT scan and a full neurological assessment after admission to hospital.

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