PULMONARY BAROTRAUMA IN DIVING

J. HOW

SYNOPSIS

Pulmonary barotrauma arises from excessive changes in pressure affecting the lungs. This condition is peculiar to the divers, aviators and chamber attendants as they experience great changes in breathing gas pressures. Four forms of pulmonary barotrauma presentations are presented. They are pulmonary tissue damage, pneumothorax, surgical emphysema and air embolism, the last being the most serious. A case of subcutaneous emphysema arising from diving is cited. Aetiology, pathology, signs, symptoms, treatment and prevention are discussed.

INTRODUCTION

Pulmonary barotrauma arises from the effects of excessive pressure causing over-distension and rupture of the lungs. This condition is widely known among the diving fraternity as "burst lung". It will result if one fails to exhale while surfacing from a compressed air or mixture dive.

Jacques-Yves Cousteau in 1942 invented the compensated demand regulator and with this, a diver could swim as deep and as free as a fish. The valve equilibrates the gas pressure breathed by the diver with the ambient water pressure surrounding the diver. If the diver then ascends without exhalation ie by holding his breath, the lungs will distend and then burst like a balloon. Under these conditions the gas obeys Boyle's Law. Boyle's Law states that "At a constant temperature, the volume of a given quantity of gas varies inversely to its absolute pressure."

Chamber attendants, Caisson workers and medical personnel doing hyperbaric work are also liable to pulmonary barotrauma. So are aviators, especially when they are drilled in "explosive decompression." Christoph in 1974 reported pulmonary barotrauma in anaesthesiologic practice.

Singapore Armed Forces
J. How, M.B., B.S., Dip. D.H.M.
Lt. Colonel

CASE REPORT: H. K. K., AGED — 26, DIVER

History

H. K. K. made a dive at 10.00 am on 20 May 76. The depth of the dive ranged from 3 metres to about 10 metres. He was swimming with a compressed air set both hands pulling very hard on the Jackstay. After an hour he went on reserve and started to surface. On surfacing he immediately felt a pain in the whole chest. He inflated his life jacket and removed the demand valve. Every breath he took became painful. On reaching shore, H. K. K. noticed a change in his voice and his neck was swollen. He noticed a "crackling" feeling in his neck. When questioned, H. K. K. replied he was not suffering from a cold before his dive. However, at 8 a.m. that morning he snorkled to 5 metres and could not "clear" his ears. He attempted twice and gave up when there was pain in his ears.

Past History

No childhood or past illnesses of significance. A pre-course physical was done on 2nd April 1976 with no abnormal findings.

Examination

General condition — Good. Rational Pulse: 74/min. BP: 120/70. Resp Rate: 18/min. Spoke with a soft low husky voice. No dyspnoea. No cyanosis. Very slight chest pain with deep inspiration. The neck was swollen.

Clinical examination showed subcutaneous emphysema in both sides of his neck and supraclavicular regions. Subcutaneous emphysema was also present in the front upper chest. The lungs were clear. The heart sounds were normal but faint. CNS examination showed no abnormality. Fundi were normal. Both tympanic membrances had Grade I aural barotrauma. X-ray revealed no pneumothorax or collapse of the lungs but subcutaneous emphysema were prominent in the neck, and there was also evidence of mediastinal emphysema. (Fig 1). ECG showed no abnormality.

Treatment

H. K. K. was immediately given 100 per cent oxygen via a free flow mask at a flow rate of 10L/min. The Recompression Chamber was meanwhile prepared and kept on standby. Oxygen 100 per cent free flow was administered for 48 hours on the following regime.

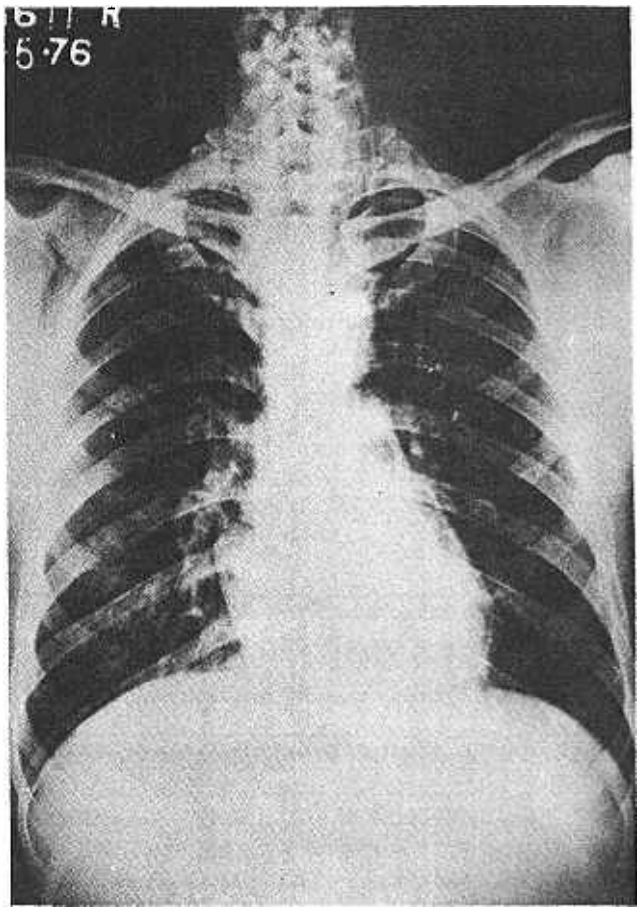

Fig. 1 Subcutaneous Emphysema prominent in the neck, with also evidence of mediastinal emphysema.

1st 12 hours — 1 hr breathing oxygen with 10 mins breathing air
2nd 12 hours — 1 hr breathing oxygen with 30 mins breathing air
Next 24 hours — 1 hr breathing oxygen alternate with 1 hr breathing air
Oxygen was taken off after 48 hours.

Progress

The Central Nervous System was normal with no indication of air embolism throughout his stay in the Sick Bay. There was no difficulty in breathing, cyanosis or any respiratory embarrassment.

There was no further chest pain within 48 hours of treatment with surface 100 per cent oxygen. The subcutaneous emphysema was present for the next 17 days. By the 5th day, the subcutaneous emphysema in the upper chest was re-absorbed. By the 17th day, the neck was entirely free from the subcutaneous emphysema. The low husky voice lasted for 12 hours. Difficulty in swallowing was experienced for 2 days.

Pulmonary Function Test by the use of vitalograph was not done on admission for fear of aggravating his condition. However, VC, FEV_1 and FEV_1 per cent was done on 14th June 1976.

Findings:	VC	=	3.35L
	FEV_1	=	2.70L
	FEV_1 per cent	=	80.5%
	Expected VC	=	4.01L

These findings showed no change from those recorded on 2nd April 1976 when he was attending a diving course.

A repeat X-ray of the chest and neck taken on 24th May 1976 showed lesser amount of air present in the subcutaneous tissue of the neck. The lungs were normal.

H. K. K. was discharged from the Sick Bay on 1st June 1976. He was suspended from diving for 6 months.

DISCUSSION

Aetiology

Breathholding while surfacing from a SCUBA dive is the commonest way of causing pulmonary barotrauma. It can occur in free-ascent practices or in submarine escape training. Of the 67,000 free ascent exercises carried out in the Escape Training Tank at New London within a five year period, 7 cases with one death (Liebow et al, 1959.) have occurred. At other times pulmonary barotrauma can result from panic where the diver fails to breathe out, in the desperate attempt to reach the water surface. The closed glottis prevents air in the lungs from escaping resulting in a build-up of pressure and volume. The thin alveolar membrances overdistend and rupture consequently.

Certain lung conditions predispose the diver to suffer from pulmonary barotrauma through the rupture of air trappings within the lungs. Such ruptures occur irrespective of whether the breath is held while surfacing from a dive. These conditions include asthma, tuberculous cavitations, lung bullae and blebs, cystic diseases of the lungs (Collins, 1962), chronic bronchitis with emphysema, tumours and calcified lymph nodes compressing on bronchi, Walder (1973) mentioned that pulmonary barotrauma is prone to occur in a diver diving with even mild conditions, like a common cold and influenza affecting the lungs.

The change of pressure causing the damage to the lungs may be as little as 80mm Hg (Collins, 1962) which is equivalent to an ascent of about 1 metre in sea water. However, it is the last 10 metres towards the surface of water which poses the greatest danger, as the variation in pressure at the shallower depths causes the greatest change in the volume of the lungs.

Pathology

Irrespective of the mode of causation, pulmonary barotrauma is manifested in 4 forms, namely pulmonary tissue damage, surgical emphysema, pneumothorax and air embolism. These conditions can occur singly or in combinations (Edmonds et al, 1976). Air embolism is rarer with pneumothorax cases and is commoner with mediastinal emphysema (Malhotra and Wright, 1961).

With a closed glottis or having some alveoli blocked by viscid mucus or tumour, a change of pressure from high to low pressure in the environment may be disastrous. Walder in 1973, noted that the pleura in man dips down between adjacent groups of alveoli to form partitions. The partitions are made up of loose tissues with a branch of the pulmonary vein running at the base of the partition. Uneven movement between adjacent sacs, as in the case when blocked alveoli overdistend with changes of pressure and volume, causes a shearing force on the partition resulting in tear of the alveoli walls and the partition with its blood vessels. Should the pleura be torn in the shearing action, then a pneumothorax accompanied by possibly a haemothorax, would result. In experiments with rabbits, Malhotra and Wright (1961) showed a pin point tear in the inferior surfaces of the middle and lower lobes. Tears were not seen in the apical lobe or the hilar structures. If the pleura is intact, air from the ruptured alveoli tracts along the interstitial planes under the pleura towards the hilum into the mediastinum and up the neck in the form of subcutanious emphysema. Post-mortem findings in animals (Malhotra & Wright, 1961) showed scattered subpleural haemorrhages over both lungs. Pneumopericardium and pneumoperitoneum are rare presentations. Air getting into the damaged blood vessels would cause air embolism.

In the case presented, the patient suffered from pulmonary barotrauma leading to subcutaneous emphysema. It is likely from the history of difficulty in clearing his ears and the finding of aural barotrauma, that he could have suffered from a cold causing part of his lungs to be obstructed resulting in rupture on surfacing from the shallow dive. However, the lung lesion was not evident on X-rays.

Signs and Symptoms

When pulmonary barotrauma appears in the form of subcutaneous emphysema around the neck and upper chest, other manifestations may be associated. These included: a change of voice, fullness of the throat, breathing and swallowing difficulties, reduction in the area of cardiac dullness, faint heart sounds or crepitus related to heart sounds (Hammon's sign) and cardiac failure.

It was fortunate for the patient that neither pneumothorax nor the more serious condition of air embolism developed. Air embolism could have resulted in unconsciousness, convulsion, blindness, paralysis, paresthesia, vertigo, confusion, skin marbling and cyanosis. Gas bubbles may be seen in retinal vessels. ECG may show ishaemic features or arrhythmass. EEGs tend towards slowing or flattening of waves. Death from coronary or cerebrovascular occlusion by bubbles is possible. The patient was carefully observed for CNS manifestations.

Treatment

Mild to moderate cases of subcutaneous emphysema from pulmonary barotrauma can be treated with 100 per cent free flow oxygen. Positive pressure respiration should be avoided, if possible. Severe cases may require recompression therapy to 20m pressure equivalent of sea-water and decompression slowly with Workman's Long Oxygen Table.

This patient was treated entirely with 100 per cent oxygen at atmospheric pressure. It must be stressed that pneumothorax and air embolism must be excluded before following this line of treatment. In doubtful cases, it woul be best to start recompression therapy immediately.

Prevention

One short-sighted way of preventing pulmonary barotrauma, is to abandon the practice of free ascent training. This drill requires the diver to ditch his diving equipment at depth and swim to the surface breathing out slowly at the start but more freely towards the surface. The practice serves to instil confidence to the diver in the event of air running out, malfunction of equipment and loss of demand valve from the mouth. Many centres have stopped this practice but others favour its continuance. Some feel strongly that many deaths through panic could be avoided through proper practice of free ascent. Recompression facilities must, however, be available when free ascents drills are carried out. An almost similar submarine escape drill is done by the submariners in huge towers and all safety features are incorporated.

It has been mentioned that even with proper diving techniques, pulmonary barotrauma could develop when the diver dives with a cold, influenza, chronic bronchitis, asthma, cystic lung diseases or tumours. In this respect, a proper medical examination before pursuing diving as a sport or profession is absolutely necessary.

A proper medical history will at most times assist the doctor in reaching a decision. A heavy smoker should be investigated thoroughly. Clinical examination of the chest is important. The presence of expiratory rhonchi, with or without a history of smoking, should give cause for suspicion and demand further investigation.

Vitalograph measurement of VC, FEV_1 and FEV_1 per cent could be used as an indicator of the degree of respiratory obstruction. An FEV_1 per cent of less than 80 per cent must be viewed with strong suspicion and with less than 75 per cent, diving should not be undertaken.

Chest X-ray could eliminate some troublesome conditions. An inspiration, expiration and lateral chest X-ray views might help to exclude blebs, bullae and cystic lung diseases. Small sub-clinical cysts are difficult to detect.

As a continuous safe guard, all divers should be medically examined yearly. The need for periodic medical review is more urgent than those in other vocations. In the four year survey of the mortality of British Divers, Crockford and Dyer in 1975 traced a fatal accident rate of 11.7 to 15.6 per 1000 men years of exposure, placing diving as the occupation with the highest risk.

As sports diving is gaining popularity and commercial diving is becoming lucrative, commercial diving schools and subaqua clubs are now shouldering great responsibility in the training of divers. The high quality of instruction and instructors will go a long way in preventing unnecessary deaths through pulmonary barotrauma.

To the knowledgeable, diving unravels a fascinating undersea world of beauty and wealth. But to the unwary and the untrained, the first step into the water may be the last.

ACKNOWLEDGEMENT

I wish to thank the Chief Medical Officer of the Singapore Armed Forces for permission to publish this article.

REFERENCES

1. Christoph, G.: Barotrauma der Lunge. Anaesth. Prax. 9:57-66; 1974.
2. Collins, J.: An Usual case of Air Embolism Percipitated by Decompression. New Eng. J. Med. 266:595-598 1962.
3. Crockford, G.W., and Dyer, D.F. (Mrs): A fair year survey of Mortality in British Divers. SPUMS Newsletter p 35 Jul-Dec 1975.
4. Edmonds, C., Lowry, C., and Pennyfather, J.: Diving and Subaquatic Medicine. A Diving Medical Centre Publications, Mosman 2088, Australia: p93-100, 1976.
5. Liebow, A.A., Stark, J.E., Vogel, J., and Schaeffer, K.E.: Intrapulmonary air trapping in submarine escape training casualties. U.S. Armed Forced M.J. 10:365-289, 1959.
6. Malhotra, M.S. and Wright, H.C.L. Arterial Air Embolism during decompression and its prevention. Proceedings of the Royal Society (Biology), 154: p 418-427, 1961.
7. Walder, D.N.: Man in the deep — Part I. Oceans 2000. Third World Congress of Underwater Activities, p 24-25, London. British Sub-Aqua Club, 1973.