SYSTEMIC AIR EMBOLISM FOLLOWING MANUAL LUNG INFLATION

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ABSTRACT

A case is reported in which a young man undergoing exploratory thoracotomy following a gunshot wound had a cardiac arrest during reinflation of the collapsed lung. Systemic air embolism was suspected as the cause. Subsequent neurological examination and C.T. scan demonstrated a lesion ascribable to this. Discontinuation of nitrous oxide, aspiration of left heart chambers and a head-down position are recommended as immediate treatment if this diagnosis is made. In cases with piercing lung trauma high inflation pressures should be avoided to prevent embolism.

KEY WORDS: COMPLICATIONS, air embolism, chest trauma.

SYSTEMIC AIR embolism has been increasingly reported as the cause of cardiac arrest in patients who have received stab or gunshot wounds to the chest. Recently we encountered such a case which forms the basis of this report.

CASE REPORT

A previously healthy 19 year old white male was admitted to the emergency room one hour after a gunshot wound to the chest. The entry wound from the 0.22 calibre bullet was three cm above and medial to the left nipple with an exit wound at the same level posteriorly. On admission the patient was shocked but conscious with a pulse rate of 130, a blood pressure of 70/40 mm Hg and a respiratory rate of 33 breaths per minute. An underwater-seal drain was inserted into the left pleural cavity and 500 ml of blood was drained. Resuscitation was started with intravenous crystalloid solution and the patient was transferred to the operating room for thoracotomy. A total volume of three litres of lactated Ringer's solution was transfused before operation. Preoperative electrolytes were normal with Hgb 11.6 and Hct 33.1.

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On arrival the patient was agitated, moderately cyanosed while breathing 40 per cent oxygen by face mask, with pulse rate of 100 min and blood pressure 100/50 mm Hg. Anaesthesia was induced with thiopentone 100 mg and succinylcholine 100 mg intravenously. The trachea was intubated with a size 9 Shiley tracheal tube. Maintenance was with nitrous oxide and oxygen two litres/min of each and pancuronium 7 mg for muscle relaxation. Fentanyl 0.05 mg was given shortly after induction and repeated once. The patient was positioned for a left thoracotomy. The right radial artery was cannulated for continuous pressure monitoring. The patient was ventilated with a tidal volume of 800 ml at a rate of 12 breaths per minute.

At thoracotomy it was found that the bullet had penetrated both the left upper and lower lobes, had partially torn the left phrenic nerve and grazed the left side of the pericardium. The phrenic artery was severed but there was no damage to the major pulmonary vessels. Twelve hundred ml of clotted blood was evacuated from the pleural cavity. Thirty minutes after induction of anaesthesia 1.5 litres of lactated Ringer's solution and three units of blood had been transfused. The pulse rate had been steady at 100 to 110/min and blood pressure at 100/60 mm Hg. After haemostasis had been achieved the surgeon requested that the partially collapsed lung be reinflated. Manual intermittent positive pressure ventilation was therefore started with the reservoir bag being held in inspiration for a few seconds with each breath to expand the atelectatic segments. The maximum airway pressures noted were 25 to 30 cm H₂O. P.E.E.P. was not

used. Immediately after the lung had been fully expanded, the blood pressure fell abruptly to zero. The pericardium was opened to facilitate direct cardiac massage. Inspection showed a full, deeply cyanosed heart in ventricular fibrillation. Calcium chloride 1 gm, epinephrine 2 ml of 1/10,000 solution and sodium bicarbonate 44.6 mmol were given intravenously. D.C. shock was applied twice. Normal sinus rhythm returned after two minutes with a blood pressure of 180/90 mm Hg and a pulse rate of 120/min. Some ST segment depression was noted on the electrocardiogram. Direct inspection of the coronary arteries failed to visualize air bubbles. Nitrous oxide was discontinued at this stage and anaesthesia was maintained with enflurane in oxygen. Mechanical I.P.P.V. was reinstituted with the same volumes and rate used before reflation. Arterial blood gases after arrest revealed $[H^+]_a$ 60.26 nmol/l (pH 7.22), P_{aCO_2} 8.8 kPa (66 mm Hg), P_{aO_2} 19.4 kPa (146 mm Hg) and bicarbonate 25.7 mmol/l. The chest was then closed without further incident. The patient was transferred to the intensive care unit, maintained on a ventilator for four hours and the trachea was extubated two hours later.

Post-operatively the patient regained consciousness fully but manifested some neurological deficit in that he appeared to have difficulty hearing. Otoscopic examination revealed no abnormality. A neurologist examined the patient 24 hours after operation and diagnosed the rare picture of fine word deafness (a bilateral superior temporal lesion) along with other dysphasic features. This diagnosis was further supported by CT scan which showed bilateral discrete temporal lobe infarcts. His overall condition improved steadily over the next 24 hours and on the third postoperative day the patient was transferred to a general ward. Two weeks after operation the neurological condition had improved considerably and the patient was discharged.

DISCUSSION

Considering the patient's age, physical status before injury and the suddenness of cardiac arrest, the most likely causative factor would seem to be pulmonary venous air embolism. The post-operative finding of a discrete neurological lesion also points to this diagnosis.

The so-called "pleural shock" which was occasionally reported during various diagnostic and therapeutic procedures on the lungs was

attributed by Wever in 1914 to aspiration of air into the systemic circulation through damaged pulmonary veins. In 1967 a case of pulmonary venous air embolism during thoracotomy was described in a man with pulmonary tuberculosis.¹ This led to experimental work in dogs which demonstrated that at inflation pressures of 40 mm Hg the normal pressure gradient between the bronchus and pulmonary venous system was reversed. At this stage incision of the lung parenchyma led to spontaneous passage of air into the left atrium, leading to ventricular fibrillation. Further work in 1978 using a similar experiment showed that at inflation pressures of 65 mm Hg in dogs, they all got air embolization, even a control group with no parenchymal damage.²

In 1973 Thomas and Roe³ reported four cases of systemic air embolism following gunshot (two cases) and stab wounds (two cases). In 1977 Graham *et al.* reported twenty-two cases of air embolism following either stab or gunshot wounds.⁴ In all cases the diagnosis was made at thoracotomy by visualizing air in the coronary arteries or by aspiration of air from the aorta or left ventricle. It was found that the most significant factor and the one which was present in all twenty-two cases was positive pressure ventilation. Further experimental work in dogs reported by Ponn, *et al.*⁵ suggested that the emboli may be small and detectable only by sophisticated means. Using an ultrasonic bubble detector placed over the carotids, air embolism was found in every case with pulmonary laceration. It was concluded that a low index of suspicion plus the fact that air bubbles have often disappeared by post mortem means that this diagnosis is often missed. Fibrotic lungs which fail to collapse down when incised are more likely to cause embolism, since they will not obliterate any fistula created.

To minimize the risks of pulmonary venous air embolism occurring, a number of steps may be taken.⁵ Hyperventilation should be avoided where possible. If this cannot be done, then clamping of the pulmonary vein during inflation will be helpful. Manual compression of a laceration site should be avoided, since this may dislodge stagnant air bubbles into the pulmonary veins. By positioning the patient so that the lacerated region lies below the level of the atria, the pulmonary venous pressure at this level would be increased and would again reduce the chance of embolization. A head down position will minimize the flow of air bubbles to the

brain. If the diagnosis has been made, cross clamping of the aorta for a short period may be helpful. Aspiration of the left heart chambers and the aortic arch during thoracotomy if air embolism is suspected may decrease the amount of air reaching the cerebral and coronary arterial beds. If nitrous oxide is being used it should be discontinued as it will diffuse into the air bubbles faster than nitrogen can leave, thus leading to an increase in the size of the bubbles.^{6,7}

Finally if all these measures are unsuccessful, a case has been reported which was treated successfully by using a hyperbaric chamber to minimize the neurological deficit caused by cerebral air emboli.⁷

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RÉSUMÉ

Les auteurs rapportent un cas d'arrêt cardiaque survenu à la suite de la réexpansion du poumon, au cours d'une thoracotomie exploratrice, chez un jeune homme ayant subi une blessure par balle. L'accident fut attribué à une embolie gazeuse, diagnostic confirmé par la suite par l'examen neurologique et la tomographie axiale cérébrale. Lorsque ce diagnostic est suspecté, on recommande de suspendre l'administration du protoxyde d'azote, d'aspirer les cavités cardiaques afin d'y évacuer l'air et de placer le malade en Trendelenbourg. Les pressions inspiratoires élevées devraient être évitées dans le but de prévenir les embolies gazeuses dans le cas de traumatismes perforants du poumon.