SYSTEMIC AIR EMBOLISM FOLLOWING INDUCTION OF ARTIFICIAL PNEUMOTHORAX UNDER ANAESTHESIA, WITH SUCCESSFUL MANAGEMENT

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SUMMARY

Systemic air embolism occurred in a patient during general anaesthesia, with positive pressure ventilation, following induction of artificial pneumothorax to assist in the diagnosis of a mediastinal mass. A sudden change in vital signs together with neurological abnormalities suggested involvement of both coronary and cerebral arteries. A trace of blood was noticed in the syringe which the surgeon had used to create the artificial pneumothorax. The patient was treated with hyperbaric oxygen and recovered satisfactorily, despite a 10-h interval between the air embolus and the institution of definitive therapy.

Air embolism may occur in the pulmonary or systemic circulations. Systemic air embolism can occur as a complication of cardiopulmonary bypass, therapeutic pneumothorax, haemodialysis and paradoxical air embolism through a patent foramen ovale. Thomas and colleagues (Thomas and Roe, 1973; Thomas and Stephen, 1974) demonstrated both experimentally and clinically that a penetrating injury to the lung may produce an acute broncho-venous fistula resulting in arterial air embolism, particularly when positive pressure ventilation is being used. Arterial air embolism from broncho-venous fistulae has also been reported from lung penetration during thoracentesis (Van Bahr, 1944) and lung biopsy (Youmans et al., 1970).

The authors encountered a similar situation in a patient who was undergoing induction of artificial pneumothorax for diagnostic purposes under general anaesthesia with positive pressure ventilation.

CASE REPORT

A 23-year-old healthy white male was admitted to hospital with a middle mediastinal mass projecting into the left lung field, detected on a routine chest x-ray. The patient was to undergo bronchoscopy oesophagoscopy and possible left thoracotomy.

Premedication consisted of pethidine 75 mg, hydroxyzine pamoate 50 mg, and atropine 0.5 mg, all administered i.m. 1 h before operation. Anaesthesia was induced with thiopentone 300 mg i.v. and maintained i.v. 1-2%, and nitrous oxide in 50% oxygen. Suxamethonium 100 mg was given to facilitate endotracheal intubation, using a cuffed Portex orotracheal tube (8.5 mm) and neuromuscular blockade was maintained with an i.v. infusion of suxamethonium. Ventilation was controlled manually.

Both fibreoptic bronchoscopy and oesophagoscopy were uneventful and uninformative.

To assess the relationship of the mediastinal mass to the surrounding structures, the surgeon induced a left artificial pneumothorax, before which nitrous oxide was discontinued. The surgeon used an 18-gauge spinal needle, a 60-ml syringe and a three-way stopcock. A trace of blood was noticed in the syringe following aspiration. After repositioning the needle, 150 ml of air was injected over 1 min into the left side of the chest in the anterior axillary line, just below the nipple.

Suddenly, the pattern of the E.C.G. changed from sinus rhythm to severe bradycardia, with intraventricular conduction defect and idioventricular rhythm. This was accompanied by a transient decrease in arterial pressure from 130/90 mm Hg to 90/50 mm Hg. Heart sounds became muffled. Patchy areas of cyanosis developed over the neck and the upper chest. Halothane and suxamethonium infusion were discontinued, but the patient developed generalized rigidity,
During the next week with an excellent neurological recovery. The patient regained consciousness on the 6th day. After 1 week, dysphasia and right hemiparesis were still present. The speech defect diminished to the point at which difficulty with finding words was apparent only when the patient was fatigued or under stress. The hemiparesis persisted for approximately 2.5 months. An intention tremor of both hands, more marked in the right, persisted for 11 months after the incident.

DISCUSSION

Systemic embolism following penetrating lung trauma has been appreciated only recently. Forlanini first advocated artificial pneumothorax for therapy of pulmonary tuberculosis (Schlaepfer, 1922). After this procedure became popular, numerous cases of "pleural shock" were reported (Van Allen, Hrdina and Clark, 1929). Some deaths occurred after the introduction of a needle into the thoracic cavity before any air was injected (Reger and Kohl, 1926), presumably because the needle had injured the lung parenchyma. In 1899 Murphy suggested that air embolism might be the cause of pleural shock (Schlaepfer, 1922). Brauder reported a case in which the pulmonary veins were shown to be the portal of entry for air embolism (Schlaepfer, 1922).

The importance of air embolism following penetrating lung trauma was not fully recognized until Thomas and others (Thomas and Roe, 1973; Thomas and Stephen, 1974), in both clinical and experimental studies, demonstrated that air could pass from the pulmonary to the systemic circulation. It is well known that the entrance of air into the pulmonary veins can be fatal by producing systemic embolism of cerebral and coronary vessels (Geoghegan and Lam, 1953).

Experimentally, cerebral air embolism is fatal when 0.5–1.5 ml kg⁻¹ of air is injected into the cerebral circulation (Fries et al., 1957). Similar experiments with coronary air embolism have shown that 0.05 ml of air injected into the left anterior descending artery or 1.5 ml into the left ventricle is fatal (Rukstinas, 1931). Relatively large amounts of air (5–8 ml kg⁻¹) may be tolerated in the venous circulation (Durant et al., 1949).

It has been falsely assumed that venous embolism is relatively harmless because the pulmonary capillaries are efficient air filters (Durant et al., 1949). However, Spencer and Oyama (1971) showed that air can cross the pulmonary capillaries when the pulmonary artery pressure exceeds 29 mm Hg. Air in the venous side can also pass to the arterial side...
through a patent foramen ovale. The frequency of patent foramen ovale is found to be as high as 25% in some autopsy series (Van Allen, Hrdina and Clark, 1929).

The principal symptoms of arterial air embolism are those resulting from involvement of the coronary, cerebral and renal arteries. The position of the patient at the time of embolization affects the distribution of emboli. Air embolism is associated with sudden changes in vital signs and usually presents with widespread clinical manifestations.

In the case reported here, the sudden changes in vital signs and failure to regain consciousness, together with the appearance of a trace of blood in the syringe on aspiration, led us to the diagnosis of air embolism. The patient showed evidence of coronary emboli, manifested by a sudden decrease in arterial pressure, muffled heart sounds and appearance of slow wide QRS complexes on the e.c.g.

Frank convulsions were not noted because the patient was paralysed, but as soon as the suxamethonium infusion was discontinued, it was extremely difficult to ventilate the lungs. The patient then started to assume a decerebrate posture with opisthotonus. The marked tachypnoea was thought to be a reflection of cerebral involvement. Failure to regain consciousness after operation was further evidence of cerebral involvement, but the fact that he improved within 20 min of initial compression treatment was diagnostic of cerebral embolism.

Artificial pneumothorax should not be created under anaesthesia, particularly with positive pressure ventilation. Embolism should be suspected if the patient shows sudden changes in vital signs. If nitrous oxide is used, it should be discontinued immediately as it can lead to an increase in the size of the air bubbles.

If air embolism occurs, the patient should be turned on to his left side in a head-down position in order to keep the bubbles of air away from the pulmonary outflow tract. An attempt should be made to aspirate the air when possible. If there is evidence of penetrating lung trauma, it is wise to avoid positive pressure ventilation whenever possible. If all these measures fail, then treatment with compression to 6–7 atm abs. in a hyperbaric chamber, followed by hyperbaric oxygen therapy, is indicated (Kinwall, 1973; Thiede and Manley, 1976). This case report confirms the fact that hyperbaric therapy may still be of use even after several hours have elapsed since the onset of symptoms.

REFERENCES

EMBOLIE GAZEUSE SYSTEMIQUE APRES INDUCTION D'UNE PNEUMOTHORAX ARTIFICIEL SOUS ANESTHESIE ET CONDUITE SATISFAISANTE DU TRAITEMENT

RESUME
Une embolie gazeuse systémique s’est produite sur un malade au cours d’une anesthésie générale, avec ventilation sous pression positive, après l’induction d’un pneumothorax artificiel devant aider au diagnostic d’une masse médiastinale. Un changement soudain dans les signes vitaux, ainsi que des anomalies neurologiques ont laissé penser à une lésion des artères coronaire et cérébrale. On a remarqué une trace de sang dans la seringue que le chirurgien avait utilisée pour créer le pneumothorax artificiel. Le malade a été soigné par oxygénothérapie hyperbare et a récupéré d’une manière satisfaisante en dépit d’un intervalle de 10 h entre l’embolie gazeuse et la mise en place d’une thérapie définitive.

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SYSTEMISCHE LUFTEMBOLIE NACH
INDUKTION VON KÜNSTLICHEM
PNEUMOTHORAX UNTER NARKOSE, MIT
ERFOLGREICHER BEHANDLUNG

ZUSAMMENFASSUNG

EMBOLIA SISTEMICA DE AIRE TRAS LA
INDUCCION DE NEUMOTORAX ARTIFICIAL
BAJO ANESTESIA, TRATADA CON EXITO

SUMARIO
Se produjo una embolia sistémica de aire en un paciente durante anestesia general, con ventilación de presión positiva, tras la inducción de neumotorax artificial para asistir en el diagnóstico de una masa mediastinal. Un cambio repentino en los signos vitales, juntamente con anormalidades neurológicas, sugirió la participación de las arterias tanto coronaria como cerebral. Se notó una traza de sangre en la jeringa que el cirujano utilizó para crear el neumotorax artificial. El paciente fue tratado con oxígeno hiperbárico y se recuperó satisfactoriamente, a pesar de un intervalo de 10 h entre la embolia de aire y el comienzo de una terapia definitiva.