Anesthesiology
73:342–345, 1990

Left Atrial Air Embolism during Intraoperative Needle Biopsy of a Deep Pulmonary Lesion

EUGENE R. WORTH, M.D.,* ROBERT J. BURTON, JR., M.D.,† RODNEY J. LANDRENEAU, M.D.,‡
G.W.N. EGGERS, JR., M.D.,§ JACK J. CURTIS, M.D.¶

Systemic air embolism is most commonly an iatrogenic complication of an invasive procedure.[1] Discovery of air embolism is often difficult due to the evanescent nature of the air bubbles.[2] We present a case of left atrial air embolism and sudden cardiovascular collapse after intraoperative Tru-Cut (Travenol Laboratories, Inc.) needle biopsy of a left upper lobe mass during open thoracotomy.

CASE REPORT

An 80-yr-old, 73-kg woman was scheduled for open left lung biopsy and possible wedge resection of a solitary pulmonary nodule. Preoperative ECG revealed a sinus rhythm, right bundle branch block, and intra-atrial conduction delay. Arterial blood gases and pulmonary functions were normal for age. Other laboratory data and coagulation tests revealed no further abnormalities.

Ninety minutes prior to surgery, 10 mg of diazepam po, 5 mg of morphine sulfate im, and 0.2 mg of glycopyrrolate im were administered. Peripheral venous and left radial artery catheters were inserted, electrodes for monitoring ECG and heart rate were attached, an automated blood pressure cuff was attached to the right arm, and oxyhemoglobin saturation (Sp_{O_2}) was monitored *via* a pulse oximeter probe placed on the left index finger. Anesthesia induction consisted of 250 mg of sodium thiopental iv and 25 mg of atracurium besylate iv. The trachea was intubated using an 8.5-mm endotracheal tube, and breath sounds were noted to be clear and equal bilaterally. Anesthesia was maintained using nitrous oxide, oxygen (50%), and isoflurane (0.5–2%) with controlled ventilation.

Flexible bronchoscopy was performed with the patient in the supine position. The patient was then turned to the right lateral decubitus position where 0.5 mg of preservative-free morphine sulfate was instilled into the subarachnoid space at the L_3–L_4 interspace using sterile technique.

Left thoracotomy and open lung biopsy were begun, and at that time the ECG was noted to be sinus rhythm, Sp_{O_2} was 100%, and end-tidal carbon dioxide ranged between 25 and 29 mmHg. Because the pulmonary lesion was deep and parahilar in location, wedge resection could not be undertaken without sacrificing the entire left upper lobe. A biopsy of the mass was done using an 18-G Tru-Cut biopsy needle. Immediately after the needle was inserted, bright red frothy blood was noted in the anesthesia breathing circuit. In the next few minutes, profound hypotension was noted that was not responsive to intravenous fluid or vasoactive drugs. Bradycardia progressing to asystole occurred, and further resuscitative measures were instituted. Steep Trendelenburg's position, discontinuation of nitrous oxide and isoflurane, 0.8 mg of atropine (in 0.4-mg boluses), and 3 mg of epinephrine iv (in 1-mg boluses) failed to produce heart rate, rhythm, or blood pressure. The pericardium was opened, and cardiac massage was instituted. Crepitus was noted in the left atrium and ventricle, and 10–15 ml of air was aspirated from the chambers. Numerous small air bubbles were noted in the distribution of the left coronary artery during the resuscitative effort. Ventricular tachycardia and ventricular fibrillation occurred and were treated with 100 mg of lidocaine hydrochloride iv and internal defibrillation. A continuous lidocaine infusion of 2 mg/min was begun, and a single bolus injection of methylprednisolone (2 g iv) was given. Blood pressure during the resuscitative effort ranged from 40–60 mmHg (mean). An intra-aortic balloon assist device was inserted in the left femoral artery. After 45 min of resuscitative efforts, the patient's cardiac rhythm and circulation were restored with the aid of the intra-aortic balloon assist device, epicardial pacing, and multiple inotropic medications. Fundoscopic examination was not performed due to patient position and difficult resuscitation.

The chest was expeditiously closed, and the patient was transferred to the thoracic intensive care unit. On admission to intensive care, she was awakening and combative. Initial arterial blood gas study in the intensive care unit revealed *p*H of 7.373, Pa_{CO_2} of 47.7 mmHg, Pa_{O_2} of 249.5 mmHg, and HCO_3 of 26.9 mEq/l. Initial ventilator settings were tidal volume of 750 cc, assist-control rate of 12 breaths per min, FI_{O_2} of 100%, and PEEP of 5 cmH_2O. Seven hours later, the patient was noted to be awake, alert, and trying to talk. She moved all extremities to command and had normal touch and pain sensation. Respiratory support was gradually withdrawn, and her trachea was extubated without difficulty.

Anteroseptal myocardial infarction was confirmed by enzyme studies and ECG on the second postoperative day. The patient was transferred from the intensive care unit on the third postoperative day. Her remaining hospital course was complicated by an episode of atrial flutter on the fifth postoperative day that responded to digoxin and quinidine. Neurologically, she showed a moderate amount of confusion during her stay in the intensive care unit; however, it cleared by the third postoperative day. On the 10th postoperative day, she was discharged from the hospital with no neurologic deficits noted and stable cardiac status. On follow-up visit, the myocardial infarction pattern was still seen on the ECG.

DISCUSSION

In a previously reported case, percutaneous thin needle aspiration biopsy in a patient with adult respiratory distress syndrome during positive-pressure ventilation in an intensive care unit led to fatal cerebral air embolism. The events immediately following the biopsy were hypotension, bradycardia, decreased cardiac output, and unre-

* Assistant Professor, Department of Anesthesiology.
† Resident, Department of Anesthesiology.
‡ Assistant Professor, Division of Cardiothoracic Surgery.
§ Professor and Chairman, Department of Anesthesiology.
¶ Professor and Director, Division of Cardiothoracic Surgery.

Received from the Department of Anesthesiology and Division of Cardiothoracic Surgery, University of Missouri Hospital and Clinics, Columbia, Missouri. Accepted for publication March 6, 1990.

Address reprint requests to Dr. Worth: Department of Anesthesiology, 3N15 Health Sciences Center, Columbia, Missouri 65212.

Key words: Complications: cardiac arrest; hypotension. Embolism: air. Surgery, thoracic.

sponsiveness.[3] In our patient, bright red blood in the anesthesia breathing circuit and sudden cardiovascular collapse were the events that heralded left atrial air embolism. We believe this to be the first reported case of left atrial air embolism complicating intraoperative needle biopsy of a lung mass during open thoracotomy under general anesthesia and positive-pressure ventilation.

We should distinguish between venous and systemic (*i.e.*, cerebral or coronary) air embolism. Fatalities from venous air embolism have been reported in the literature.[4,5] In the cases reported, the rate of injection is more important than the total volume infused.[4,5] The mechanism by which air injected into the venous circulation enters the arterial circulation remains controversial. It has been shown that air can shunt from the right to the left heart through anatomic defects, either atrial septal defect or patent foramen ovale,[6,7] but paradoxical air embolism has been reported in the absence of intracardiac septal defects.[8,9] Under certain conditions, venous air can pass through the pulmonary circulation to the arterial side *via* physiological shunts.[10] Venous infusions of air at 0.35 ml/kg/min have been shown to result in systemic embolization in the dog with an intact interatrial septum.[11] However, very small amounts of air introduced into the pulmonary veins can be fatal. As little as 0.5 to 1 ml of air injected into a pulmonary vein can cause cardiac arrest from coronary air embolus and focal coronary ischemia.[12] Two or three milliliters of air injected into the cerebral circulation has been fatal.[7,12]

Percutaneous thin needle biopsy in the awake, closed-chest, spontaneously breathing patient is a relatively safe, effective, and commonly practiced means to diagnose a wide range of pulmonary pathology. Minor complications such as pneumothorax, hemoptysis, and parenchymal bleeding are common, but reported fatalities are rare.[12–17] Only seven cases of systemic air embolism have been reported as a complication of this procedure, five of which were fatal.[1,12,15]

There are three possible mechanisms whereby air can enter the systemic circulation during needle biopsy of the chest. First, air introduced into the venous side of the circulation may traverse various pathways to the arterial side. Secondly, the tip of the needle may lie in the pulmonary vein. If atmospheric pressure exceeds pulmonary vein pressure with the needle open to the atmosphere, air can enter the vein (*e.g.*, rapid inspiration). Finally, the needle may pierce a bronchus, tumor, cavity, or cyst, and the nearby pulmonary vein. If the lung tissue is rigid, thus causing the injured vein to remain open, air can pass from the lung into the pulmonary vein (*e.g.*, during Valsalva's maneuver, coughing, or positive-pressure ventilation).[15]

Most, if not all, cases of fatal air embolism following needle puncture of the lung have occurred in areas of advanced consolidation, tumor, or abscess formation.[14,15,18] The potential for air to enter the systemic circulation *via* the pulmonary veins exists whenever there are openings in both the bronchial and pulmonary venous system.[18,19] We postulate that the third mechanism is the most likely cause of air embolism in our patient. A bronchial-pulmonary venous fistula was created by the placement of the Tru-Cut biopsy needle through firm tumor tissue (fig. 1). The biopsy needle traversed both the bronchus and pulmonary vein in its course through the tumor. Positive-pressure ventilation in this setting forms an artificial conduit between the bronchus and pulmonary vein during the ventilatory cycle. During the expiratory phase, pulmonary venous pressure is higher than intrabronchial pressure, allowing blood to cross the fistula appearing in the endotracheal tube and breathing circuit. During the inspiratory phase, air crosses the fistula passing into the pulmonary vein and left atrium.

Treatment for venous and systemic air embolism are also different. Immediate therapy for venous air embolism includes left lateral decubitus position and steep Trendelenburg's position (Durant's maneuver), 100% inspired oxygen, aspiration of the air (if possible) through a central venous catheter, pharmacologic and mechanical resuscitation as required, and isolating the embolic source.[16,18]

Treatment for systemic embolism, once diagnosed, depends on the end-organ involved. Coronary air embolism may cause electrocardiographic evidence of ischemia, arrhythmias, depression of myocardial function, myocardial infarction, and sudden death.[18,20–22] Our patient showed evidence of myocardial infarction on the first postoperative day. She also suffered malignant arrhythmias during the operative procedure while air was seen in the left coronary artery. Therapy involves restoring myocardial contractility, removing air when possible, and immediate hyperbaric oxygen therapy.[21,22] Hyperbaric oxygen therapy has been used to reduce the size of the bubbles in the peripheral circulation, increase oxygenation to the tissues, and possibly reduce neurologic sequelae postoperatively.[8,**,18,21,23] Inotropic agents that raise the mean aortic pressure, especially those raising diastolic blood pressure, would increase perfusion through the partially obstructed coronary arteries.[18,20,22] Maintaining a high left atrial pressure is also desirable.[19]

Cerebral air embolism is more difficult to diagnose. Manifestations of cerebral air embolism depend on the exact location of the arterial occlusion and the volume of gas disseminated in the brain.[23] Cerebral air embolism may be detected by CT scan, although, in some cases, air emboli are not readily visible immediately after the suspected incident.[2,15] Immediate hyperbaric oxygen therapy

** Pierce EC: Cerebral gas embolism (arterial) with special reference to iatrogenic accidents. Hyperbaric Oxygen Review 1:161–184, 1980.

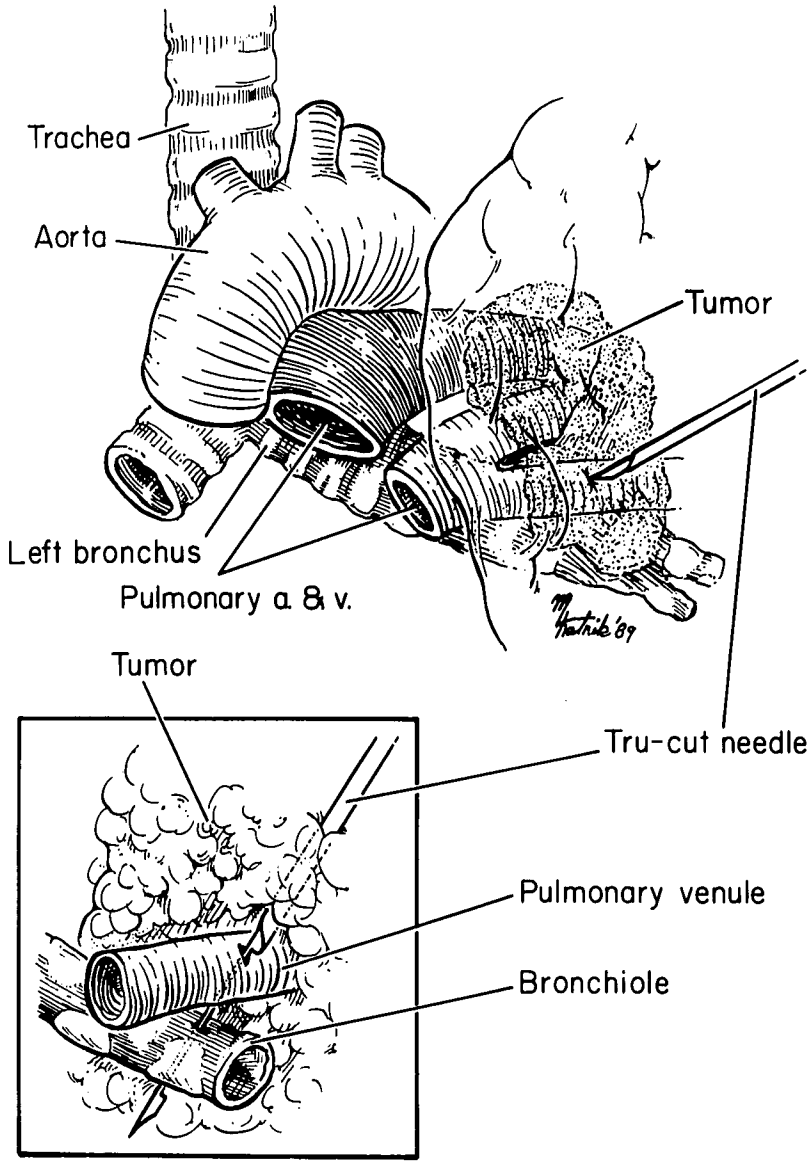

FIG. 1. Anatomic position of a deep, central lung tumor. The inset shows the path of the biopsy needle through the tumor mass. Note the proximity of the pulmonary venule and bronchiole in the needle path.

has an excellent chance of reducing the amount of air in the tissue and reducing the neurologic deficits.[15,23] Treatment also includes the use of anticonvulsants to control seizures; a large, single steroid dose for mitigation of cerebral edema; and medications to antagonize platelet aggregation.** In cases of cerebral air embolism, raising the blood pressure may cause deterioration of brain function and may worsen blood flow in the injured areas.[24] Our patient had no obvious neurologic sequelae. She was in the right lateral decubitus position and was rapidly placed into Trendelenburg's position after the onset of hypotension. In the head-down position, bubbles in the aorta are less likely to enter the cerebral circulation, thus reducing the risk of cerebral air emboli.[15,19] Reports suggest that air emboli entering the cerebral circulation in this position are distributed to neurologically silent areas of the brain.[8]

Prevention of systemic air embolism during needle biopsy of deep, central lesions of the lung is of utmost importance. The biopsy should be obtained in a quiet, collapsed lung. The lesion should be effaced as much as possible so that the needle can pass through the least amount of lung tissue. Use of a double-lumen endotracheal tube may be beneficial in reducing the pressure gradient between airway and pulmonary vein,[25] and helping to identify the firm tumor tissue. Coughing, straining, or positive-pressure ventilation should be avoided during the actual biopsy of the mass. If air embolism is noted, collapsing the affected lung with a double-lumen endotracheal tube may diminish the amount of air escaping into the systemic

circulation until the surgeon can clamp the hilar vessels and isolate the defect.[18,19]

In summary, we have described a case of left atrial air embolism during needle biopsy of a deep pulmonary mass. Rapid diagnosis and therapy are necessary to preserve circulation and prevent neurologic sequelae.

References

1. Menkin M, Schwartzman RJ: Cerebral air embolism. Arch Neurol 34:168–170, 1977
2. Jensen ME, Lipper MH: CT in iatrogenic cerebral air embolism. AJNR 7:823–827, 1986
3. Baker BK, Awwad EE: Computed tomography of a fatal cerebral air embolism following percutaneous aspiration biopsy of the lung. J Comput Assist Tomogr 12:1082–1083, 1988
4. Taylor JD: Post-mortem diagnosis of air embolism by radiography. Br Med J 1:890–892, 1952
5. Hartveit F, Lystad H, Minken A: The pathology of venous air embolism. Br J Exp Pathol 49:81–86, 1968
6. Gronert GA, Messick JM, Cucchiara RF, Michenfelder JD: Paradoxical air embolism from a patent foramen ovale. ANESTHESIOLOGY 50:548–549, 1979
7. Michel L, Poskanzer DC, McKusick KA, Nardi GL, Malt RA: Fatal paradoxical air embolism to the brain: Complication of central venous catheterization. JPEN 6:68–70, 1982
8. Kearns PJ, Haulk AA, McDonald TW: Homonymous hemianopsia due to cerebral embolism from central venous catheters. West J Med 140:615–617, 1984
9. Marquez J, Sladen A, Gendell H, Boehnke M, Mendelow H: Paradoxical cerebral air embolism without an intracardaic septal defect. J Neurosurg 55: 997–1000, 1981
10. Butler BD, Hills BA: The lung as a filter for microbubbles. J Appl Physiol 47:537–543, 1979
11. Butler BD, Hills BA: Paradoxical air embolism: Transcapillary route (letter). Crit Care Med 11:837, 1983
12. Sinner WN: Complications of percutaneous transthoracic needle aspiration biopsy. Acta Radiol Diagnosis 17:813–828, 1976
13. Lalli AF, McCormack LJ, Zelch M, Reich NE, Belovich D: Aspiration biopsies of chest lesions. Radiology 127:35–40, 1978
14. Westcott JL: Air embolism complicating percutaneous needle biopsy of the lung. Chest 63:108–110, 1973
15. Aberle DR, Gamsu G, Golden JA: Fatal systemic arterial air embolism following lung needle aspiration. Radiology 165:351–353, 1987
16. Cianci P, Posin JP, Shimshak RR, Singzon J: Air embolism complicating percutaneous thin needle biopsy of the lung. Chest 92:749–751, 1987
17. Tolly TL, Feldmeier JE, Czarnecki D: Air embolism complicating percutaneous lung biopsy. AJR 150:555–556, 1988
18. Thomas AN, Roe BB: Air embolism following penetrating lung injuries. J Thorac Cardiovasc Surg 66:533–540, 1973
19. Ponn RB, Zatarain G, Gerzberg L, Hottinger CF, Haase W, Nelsen TS: Systemic air embolism in experimental penetrating lung injuries. J Thorac Cardiovasc Surg 74:766–773, 1977
20. Oka Y, Inoue T, Hong Y, Sisto DA, Strom JA, Frater RW: Retained intracardiac air: Transesophageal echocardiography for definition of incidence and monitoring removal by improved techniques. J Thorac Cardiovasc Surg 91:329–338, 1986
21. Amar R, Halon DA, Lewis BS: Myocardial damage following coronary air embolism during coronary angiography. Cathet Cardiovasc Diagn 13:39–41, 1987
22. Hadjimiltiades S, Goldbaum TS, Mostel E, Mintz GS, Kimbiris D: Coronary air embolism during coronary angioplasty. Cathet Cardiovasc Diagn 16:164–167, 1989
23. Halpern P, Greenstein A, Melamed Y, Taitelman U, Sznajder I, Zveibil F: Arterial air embolism after penetrating lung injury. Crit Care Med 11:392–393, 1983
24. Dutka AJ, Hallenbeck JM, Kochanek P: A brief episode of severe arterial hypertension induces delayed deterioration of brain function and worsens blood flow after transient multifocal cerebral ischemia. Stroke 18:386–395, 1987
25. King MW, Aitchison JM, Nel JP: Fatal air embolism following penetrating lung trauma: An autopsy study. J Trauma 24:753–755, 1984

Anesthesiology
73:345–347, 1990

Life-threatening Apnea Following Spinal Anesthesia in Former Premature Infants

ROBIN G. COX, M.B., B.S., M.R.C.P.(U.K.), F.F.A.R.C.S.(Engl), F.R.C.P.(C),*
GERALD V. GORESKY, M.D., F.R.C.P.(C)†

With improvements in neonatal care, an increasing number of former premature infants are presenting for surgery, particularly inguinal hernia repair. Over the past decade, there have been many reports of postoperative apnea in such patients.[1–3] Several strategies have been employed to overcome this problem. These include postponing surgery for as long as possible, postoperative monitoring, use of naloxone,[4] perioperative intravenous caffeine,[5] and regional techniques such as spinal anesthesia.[6–8] It has been suggested that spinal anesthesia results in a lower incidence of postoperative apnea in former premature infants. We report two cases of life-threatening apnea following spinal anesthesia in former premature infants.

* Clinical Assistant Professor of Anaesthesia and Paediatrics.
† Associate Professor of Anaesthesia and Paediatrics.

Received from the Department of Anesthesia, University of Calgary, Alberta Children's Hospital, Calgary, Alberta, Canada. Accepted for publication March 9, 1990.

Address reprint requests to Dr. Cox: Department of Anesthesia, University of Calgary, Alberta Children's Hospital, 1820 Richmond Road S.W., Calgary, Alberta, Canada, T2J 5C7.

Key words: Anesthetic techniques: regional, spinal. Complications, postoperative: apnea. Prematurity.