Pulmonary Air Embolism During Insertion of a Permanent Transvenous Cardiac Pacemaker

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SUMMARY

In two patients pulmonary air embolism was a complication of the implantation of a permanent transvenous cardiac pacemaker. One of these patients, in whom air embolism was documented by cineradiograms, is described in detail. Cineradiograms demonstrated air in the right ventricle and main pulmonary artery with clear visualization of the opening and closing of the pulmonary valve. Both patients were treated conservatively by use of the left lateral decubitus position and administration of oxygen. The pathophysiology, hazards, and therapy of this complication are reviewed. Massive pulmonary air embolism is potentially fatal, and special care with regard to insertion of the electrode catheter should be taken in order to prevent it.

Additional Indexing Words:
Heart block Hazard of operations on the neck Cardiac resuscitation

During recent years permanent implantation of a transvenous cardiac pacemaker has become an effective method of treating patients with heart block and Stokes-Adams seizures.¹ The bipolar pacing catheter is inserted into the jugular venous system and attached to a pulse generator placed subcutaneously below the clavicle.¹ To date, this procedure has been performed at the Duke University Medical Center in 55 patients. Two of these patients experienced during the insertion and manipulation of the electrode catheter the unusual and potentially fatal complication of pulmonary air embolism. The internal jugular vein was the site of entry of air in one patient; the external jugular vein in the other. The purpose of this report is to describe in detail the complication in one of these patients in whom air embolism was documented by cineradiograms, and to review the pathophysiology, hazards, and therapy of this complication.

Report of Case

An 88-year-old white woman was admitted to the Duke University Medical Center for evaluation and therapy of a syncopal episode. She had a history of chronic pulmonary disease and bronchiectasis with many years of cough, shortness of breath, and intermittent hemoptysis. She was taking digoxin and quinidine sulfate because of a "heart attack" several years previously. Physical examination revealed a blood pressure of 100/70 mm Hg and an irregular pulse of 88 beats per minute. There were inspiratory rales and rhonchi at the left posterior lung base. The heart was not enlarged. A grade II/VI systolic ejection murmur was present at the cardiac base. Serial electrocardiograms demonstrated an intermittent nodal rhythm with subsequent progression to ventricular tachycardia and ventricular standstill. Successful resuscitation was accomplished and a temporary pacing catheter was inserted via an antecubital vein into the right ventricle.

Ten days later it was decided to insert a Cardack-Greatbatch permanent transvenous cardiac

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pacemaker. The external jugular vein was exposed surgically, and the electrode catheter was inserted via a small venotomy. It was thought that the vein was firmly secured to the catheter by a silk suture. The patient had several coughing paroxysms during the procedure. During the television monitoring of the manipulation of the electrode catheter, air was noticed in the right ventricular outflow tract and main pulmonary artery. The opening and closing of the pulmonary valve could be clearly visualized. Cinefluorograms were obtained (fig. 1). The patient was immediately placed in the left lateral decubitus position and 100% oxygen was administered by mask. Vital signs remained stable, and examination of the heart and lungs showed no abnormality. Arterial pressure monitored by a Cournand needle in the right radial artery was stable. Electrocardiograms showed no axis shift. Repeat fluoroscopy and cinefluorograms in 1 hour showed disappearance of the air bolus from the right heart. The patient remained in the left lateral decubitus position and breathed 100% oxygen for 3 hours.

The following day the patient experienced left pleuritic chest pain and a temperature of 39 C. On the subsequent day a loud leathery pleural friction rub was heard at the left lateral and posterior lung base. Moderate elevation of serum lactic dehydrogenase in combination with normal serum glutamic oxalacetic transaminase was consistent with pulmonary infarction. These symptoms and signs cleared spontaneously after 72 hours, and the patient was discharged without having any further difficulty.

Discussion

Accidental venous air embolism with the introduction of large volumes of air into the lesser circulation has complicated various medical and surgical procedures including positive pressure transfusions, hysterosalpingography, presacral air injections, pneumoencephalography, and surgery of the head and neck. Berger and associates described two cases of air embolism in 320 cases in which scalene node biopsies were performed. The present report is the first description of pulmonary air embolism as a complication of the insertion of a permanent transvenous pacemaker.

The fluctuation in intrathoracic pressures during respiration is transmitted to the large veins in the neck and can produce a sucking force during inspiration. The inspiratory negative pressure can be exaggerated in anxious persons or in patients with chronic lung disease and frequent paroxysms of coughing. Furthermore, in many individuals normal venous valves are absent in the internal and external jugular veins, which may enhance the likelihood of aspiration of large volumes of air.
Examination of the heart frequently reveals a precordial churning sound called a "millwheel" murmur, which represents the right ventricle beating against the bubbles of air. The murmur is often followed by cardiovascular collapse. This physical finding was not detected in our patients.

With the patient supine, a bolus of air in the venous system usually migrates to the right ventricular outflow tract, the highest point in the closed venous system. Early investigators believed that such a bolus of air produced obstruction of the outflow tract causing acute right heart failure. More recent work, however, suggests that cor pulmonale results when large amounts of air become impacted in the pulmonary arterioles and capillaries. The cineroentgenograms from our patient showed blood being ejected through the air without apparent obstruction at the outflow tract. When such a patient is placed in the left lateral decubitus position, the air bubble is free to move to the apex of the right ventricle. The displaced air is then churned up with blood and reaches the lungs at a much slower rate.

The hemodynamic and pathological alterations resulting from pulmonary air embolism have been studied extensively in dogs. Mandelbaum and King have shown that as little as 1.5 cc of air per kg of body weight can produce significant pulmonary hypertension, decreased cardiac output, increased right atrial pressure, and increased systolic pressure. These changes were not altered by autonomic blockade. Eighty per cent of their animals exhibited pulmonary pathology which varied from small subpleural ecchymotic areas to frank pulmonary infarction. These same investigators noted that small air emboli may pass through the lungs to the pulmonary veins and into the systemic circulation. Marchanal and co-workers concluded from studies in dogs that, in addition to pulmonary capillary obstruction by air, the "froth" secondary to the churning of blood with air distended the right ventricle and atrium, causing incompetence of the pulmonary and tricuspid valves. Although they did not observe air in the pulmonary veins, they noted that air may enter the left ventricle in a retrograde manner via the coronary sinus and thebesian veins. They also speculated that systemic embolization was possible in man through a patent foramen ovale.

The amount of air in the lesser circulation which is capable of producing death is variable and depends on its rate of introduction. In dogs, 7.5 cc of air per kg of body weight, injected rapidly, is sufficient to cause sudden death. Gottlieb and associates concluded, from reviewing case reports, that as little as 300 cc of air in the venous system can be lethal for a human. On the other hand, the minimum uniformly lethal volume of air entering the systemic circulation of a dog was found to be only 1.5 cc per kg of body weight.

In 1964, Ericsson and associates reviewed the literature and found 93 cases of venous air embolism and stressed the danger of sudden cardiovascular collapse. In 40 untreated patients, there were 37 deaths (93%), whereas treatment with the left lateral decubitus position, oxygen, and vasopressors reduced the mortality in 27 patients to 33%. Other forms of therapy that might be used with more seriously ill patients include hyperbaric oxygenation, aspiration of air from the heart, and open- and closed-chest cardiac massage. Conservative management consisting of use of the left lateral decubitus position and oxygen administration was employed for both of our patients, and more vigorous treatment was not required.

Permanent transvenous cardiac pacemakers have been utilized more and more frequently in recent years for the treatment of heart block. That pulmonary air embolism is a possible complication of the insertion of a transvenous pacemaker should be recognized. Special care with regard to catheter insertion and manipulation should be taken in order to prevent this complication, especially in patients with coexisting pulmonary disease, in whom coughing may increase the likelihood of venous air embolism. Massive pulmonary air embolism is a possible complication of the insertion of a transvenous pacemaker, and special care is essential to prevent this complication.
PULMONARY AIR EMBOLISM

air embolism is potentially fatal and should be recognized and treated promptly.

References

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