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Volume of Air in a Lethal Venous Air Embolism

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THE morbidity and mortality rates from venous air embolism is determined by the volume of air entrained, the rate of entrainment, and the position and the cardiac status of the patient. As early as 1809, Nysten[1] estimated the lethal dose of air to be 40–50 ml in a small dog and 100–120 ml in a large dog. The exact amount, 7.5 ml/kg, however, was not determined in dogs until 1953 by Oppenheimer *et al.*[2] In 1963, Munson *et al.*[3] demonstrated a lethal volume of only 0.55 ml/kg in rabbits. The lethal volume of air in an adult human is unknown but is estimated to range from 200 to 300 ml. These numbers are derived from the cases of fatalities reported by Martland,[4] Yeakel,[5] and Flanagan.[6] We report herein a case of

Case Report

A 71-yr-old, 68 kg, 173-cm man was admitted to the hospital because of congestive heart failure. Treatment included administration of diuretics and digitalis. Physical examination showed that the patient had cardiomegaly, hepatomegaly, ascites, and pitting edema. A chest radiograph confirmed marked cardiomegaly and a heart scan showed pericardial effusion. In the radiology suite, the patient was placed in a semi-Fowler position. Standard monitoring of electrocardiography (ECG) and blood pressure was used. A subxyphoid pericardiocentesis was performed using a No. 14 Jelco intracath, yielding 175 ml serosanguinous fluid with a specific gravity of 1.015, an erythrocyte count of 74,000/mm^3, and 145 leukocytes/mm^3. Results of the culture and cytology studies of the fluid were negative. Five days later, repeat chest radiography showed reaccumulation of pericardial fluid. Repeat pericardiocentesis was attempted and abandoned after the withdrawal of 75 ml bloody fluid. Pericardiocentesis was performed again 5 days later. Four hundred sixty milliliters of bloody fluid was removed and 250–300 ml air were injected through the catheter for pneumopericardiography. A week later, repeat pericardiocentesis was performed in the radiology suite. Bloody fluid, 450 ml, was aspirated without difficulty. For pneumopericardiography, 200 ml air was injected over 3–5 s through the catheter without difficulty. However, almost immediately, the patient became restless, apneic, and opisthotonic. ECG showed marked depression of ST segments with varying degrees of atrioventricular block. The procedure was discontinued. The patient was intubated and external cardiac massage was started. Resuscitation was unsuccessful and the patient died.

Autopsy was performed 3 h after death. There were no postmortem changes in organs or tissue. Because of the possibility of air embolism, water was placed between the skin flap and chest wall in the axillary region. No air escaped from either the right or the left pleura cavity. The pericardial cavity contained no air. There were 800 ml blood and several large blood clots in the pericardial cavity. Extensive fibrinous adhesions existed between the pericardium and the epicardium. The right ventricle was distended with air. There were two fresh and three healing 1-mm slits grouped in a 1-cm radius in the serosal surface of the anterior myocardium of the right ventricle corresponding to the slit in the pericardium. The pericardial cavity was filled with water. When a hole was made in the anterior aspect of the pulmonary artery 1 cm above the pulmonary valve, air bubbles escaped. Both the right and the left ventricles were moderately enlarged because of hypertrophy. The foramen ovale was probe patent but was closed with a flap-like septum primum. Tricuspid valves were normal. No air was present on the left side of heart or the coronary arteries. Further dissection of the fixed gross specimen showed again needle-sized perforations on the external and internal surfaces of the right ventricle. There was a sinus tract through the myocardium, connecting the two perforations. Both lungs were emphysematous. No other specific lesions were recognized. The other organs, including the brain, were grossly and microscopically normal.

Discussion

The minimum volume of air lethal to human beings has not been established. Martland[4] reported two fatal cases of venous air embolism that occurred during vaginal powder insufflation treatment for trichomonas infections. The total volume of air was estimated to be approximately 300 ml because six compressions of the insufflator bulb, the same number of compressions of the bulb used in the treatments, displaced 300 ml water. In the reported cases, both veins of the broad ligaments, the inferior vena cava, and the right side of the heart were distended and contained numerous air bubbles. Yeakel[5] reported a case of lethal air embolism that occurred during blood transfusion *via* a pressurized, plastic, blood container. The exact amount of entrapped air in the bag that was forced into the patient's vein is unknown, but was estimated to be approximately 200 ml, based on retrospective experiments. Flannagan *et al.*[6] reported a case of lethal complication of air embolism as a result of subclavian venipuncture. During Intracath insertion through a 14-gauge needle while the patient was in the semi-Fowler position, a rush of air was heard at the needle and the patient died within 5 min. To duplicate the clinical situation, the authors conducted an animal experiment. Based on their experiment, they found the volume flow rate of air able to be passed through a 14-gauge needle to be approximately 100 ml/s. Because it took only 1 to 2 s for insertion of the Intracath after the removal of the syringe from the needle, they estimated the

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volume of air to be approximately 200 ml. Thus, all previous case reports are conjecture or attempts at an estimate of the volume of air that was introduced intravenously. In these reported cases, air entered through peripheral veins. Air bubbles were noted along the venous channels leading to the right side of the heart. The exact amount of air that caused the patient's death is probably less than the estimated amount.

The case presented herein is unique in that a known volume of air was introduced directly into the right ventricle, resulting in the death of the patient. Because no air was found in the pericardial cavity, one can assume that all air must have been injected directly into the right ventricle. Microscopically, no air bubbles were seen in the pulmonary vascular beds, coronary arteries, or cerebral arteries to indicate paradoxical air embolization. Only the right ventricle and pulmonary artery were distended and contained air bubbles. The proximate cause of death for this patient, therefore, is most likely a result of acute right ventricular outflow tract obstruction.

An important question relates to the effects of the patient's cardiac disease on the volume of air necessary to be considered lethal. Overdistension of the right ventricle and obstruction to pulmonary blood flow are the primary pathophysiologic causes of death as a result of venous air embolism. It is possible that any impaired cardiac contractility in this patient may have decreased the volume of air necessary to produce cardiac arrest. Therefore, the lethal volume of air may be greater in adults with normal cardiac function.

In summary, estimates of 200–300 ml air have been reported to be lethal. This is the first report in an adult human to document an exact lethal volume of air, 200 ml (albeit in a patient with congestive heart failure), rather than to estimate retrospectively the lethal volume after the incident of fatal venous air embolism. It is still unclear whether this amount, 200 ml, represents the minimum volume of air considered to be lethal to healthy adult humans.

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