Treating Massive Air Embolism

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Abstract

The purpose of this paper is to present concepts regarding the physiology and treatment of massive air embolism resulting from cardiac surgery and cardiopulmonary bypass. This includes physical, pharmacological, transport, and hyperbaric measures. The concepts and review of the literature are presented to aid the cardiovascular surgical team in formulating a protocol for handling and treating air embolism.

Introduction

Of the many potential complications of cardiac surgery, one of the most dangerous and life-threatening is massive cerebral air embolism. Air emboli may be introduced into systemic arterial circulation either from the pump-oxygenator system or from the left heart following cardiotomy. In some cases the occurrence of an air embolism may be immediately evident, but it should also be suspected in a patient who demonstrates post-operative neurological deficits. The purpose of this paper is to serve as a review and summary of the available literature dealing with the pathophysiology and treatment of air embolism secondary to cardiac surgery.

Pathophysiology

The clinical picture of cerebral air embolism presents itself in three basic forms. First is the patient who does not fully regain consciousness following surgery. The second is that of the patient who regains consciousness in the early post-operative period but within 12 to 24 hours displays a decrease in sensorium, often accompanied by convulsions. Focal neurological deficits such as hemiplegia, aphasia, blindness, etc. constitute the third type of clinical picture.

The pathology of cerebral air embolism is a result of mechanical obstruction to the flow of blood, the air-blood interface, and the air-vessel wall interface. Gas bubbles that enter systemic circulation may remain lodged in the arterial circulation causing sludging of blood beyond the obstruction, or may be passed into the venous circulation. The air-blood interface is characterized by the development of a protein layer on which adhesions of platelets and lipid droplets form. This layer is often capable of separating from the original bubble surface producing further emboli. The blood also reacts to the bubble as a foreign substance triggering the reaction of leukocytes and changes in circulating platelets. The air-vessel wall interface produces a local vasospasm which may last for several minutes and increase the degree of mechanical obstruction. The presence of pressurized air in the vessel may produce pressure damage to the walls, and may act as a chemical irritant producing endothelial damage and subsequent fibrin formation. The results of these
mechanisms are cerebral hypoxia, edema, inflammation, and thrombus formation.

Treatment

The treatment of air embolism is aimed at minimizing the travel of the air to the cerebral circulation, relieving the obstruction, reducing cerebral edema, improving oxygenation of the affected area, and eliminating the nitrogen from the blood. Stoney et al. presents several mechanical measures that can be taken to remove air should a massive embolism occur in the ascending aorta. These include aortotomy, massage of the heart and great vessels, and gentle retrograde perfusion into the superior vena cava. Once cardiopulmonary bypass can be initiated, moderate to profound hypothermia should be induced to reduce the effects of cerebral edema and hypoxia. Steroids should be administered to reduce cerebral edema. However, some authors have suggested that if hyperbaric treatment is to be used, the administration of the steroids should be delayed until the patient reaches a decompression level of 1.9 atmospheres (ATA) as steroids are known to potentiate oxygen toxicity. Heparinization is useful in decreasing both the platelet adherence to the bubble surfaces and the coagulation of stagnant blood. Once embolization has occurred however, the administration of heparin probably will not be helpful since it will not reach the blocked microcirculation. Osmotic diuretics, such as urea or Mannitol should be used to reduce cerebral edema by decreasing total body water. The patient should be placed in Trendelenburg’s position to mechanically reduce bubble embolization to the cerebral circulation. If nitrous oxide anesthesia is being used, it should be immediately discontinued as it may diffuse into the bubbles increasing their size or pressure. The patient should be ventilated with an FiO₂ of 1 to aide in elimination of nitrogen from the blood. If the patient is not anesthetized sedation may be necessary to reduce convulsions and prevent shivering during induction of hypothermia.

The aspect of treatment that has made the greatest impact on the mortality of massive air embolism is hyperbaric oxygenation. The use of this modality of treatment in the post-cardiac surgery patient was first reported by Takita in 1968. Basically, the patient is placed in a chamber which is pressurized, maintained at that pressure for a short period of time, and then is depressurized at a controlled rate. The concept of the hyperbaric chamber was developed in the 1930’s for use in submarine escape training to treat lung ruptures and decompression sickness. In 1945 the Navy developed tables for regulating decompression time, but the table used for patients with central nervous system involvement required the patient and attending personnel to remain in the chamber for 38 hours. This amount of time was impractical for the post-surgical patient as much of the necessary care could not be carried out except in very large and sophisticated chambers. Also the amount of time was very taxing on the attending personnel in the chamber. Therefore, the tables were modified in 1966 to a protocol for treating air embolism that requires only 5 to 6 hours in the chamber.

Hyperbaric treatment should be considered in any case of evident or suspected air embolism, even if there would be a delay of several hours before treatment is initiated. The cardiac team should become aware of the location of a hyperbaric chamber nearest them that is large enough to handle a surgical patient and can be pressurized to a depth of 165 feet (6 ATA). When investigating various modes of transportation to a chamber, it should be remembered that travel in an unpressurized aircraft is particularly hazardous to a patient with air embolism. Prior to entering the chamber, the patient should be evaluated by a physician familiar with hyperbaric treatment to establish baseline parameters and detect possible contraindications.

After the patient enters the chamber it is rapidly pressurized to six atmospheres which applies Boyle’s law (gas volume is inversely proportional to pressure) and compresses the bubbles to approximately 17% of their original volume. Compression above six atmospheres offers no advantage in that it results in only a small change in bubble volume and may potentiate the development of nitrogen narcosis in chamber personnel. The pressure of 6 ATA is maintained for 30 minutes and then a very controlled decompression is initiated (generally over a 4 or 5 hour period).

Because mechanical compression of the bubbles cannot totally resolve emboli, 100% oxygen at hyperbaric pressures is used for three important effects. Fick’s law is applied, providing an increased concentration gradient to promote the denitrogenation of the blood. Hyperbaric oxygen also has a vasoconstrictive effect which aides in reducing intra-cranial pressure. The third effect is the preservation of the viability of affected brain tissue. At 3 ATA on 100% oxygen, the patient’s PaO₂ may exceed 2000 torr producing a large
blood-tissue concentration gradient and enhancing diffusion distance.⁸

There are certain potential dangers to hyperbaric oxygenation. One is additional nitrogen bubbles in the blood which may cause decompression sickness if the patient or chamber personnel are breathing air. This may be reduced by following strict decompression protocols and some centers use a helium-oxygen breathing mixture when at high pressures. Another potential danger is oxygen toxicity with its neurological and pulmonary reactions, although this can be avoided by not administering pure oxygen above 3 ATA of pressure.⁸ Hypoventilation caused by decreased volume of gas required for oxygenation and the increased workload of breathing (due to the denser gas) should be corrected to prevent hypercapnia and atelectasis.

Summary

The treatment of massive air embolism consists of hypothermia, steroids, heparinization, sedation, diuretics, positioning, and hyperbaric oxygenation. The goals of treatment are to restore circulation, reduce edema, reduce disturbances of clotting, protect viable tissue, and eliminate the nitrogen from the blood. With the cardiac surgical patient there is a great potential for air embolism, therefore the surgical team should be aware of the various aspects of treatment. Regarding hyperbaric treatment, the team should contact a facility and plan for the often complicated transportation and treatment of the air embolism patient. If an embolism event does occur, the chamber team should be contacted as early as possible so that chamber preparations can be started. With a concerted effort, even this dangerous complication of cardiac surgery can be treated effectively and efficiently to give the patient the best chance at recovering with little or no neurological deficit.

References