Hyperbaric Oxygenation for Arterial Air Embolism During Cardiopulmonary Bypass

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The incidence of systemic air embolism during cardiopulmonary bypass is estimated to be 0.1%. However, the vast majority of instances are unreported and quietly ignored. The result may be disability or death. The control of air embolism obviously lies in prevention. The definite and specific treatment of this complication is hyperbaric oxygen. We report 6 patients referred to our institute because of air embolism during cardiopulmonary bypass. Of the 4 patients in whom hyperbaric oxygen therapy was delayed for 17 to 20 hours, 2 showed partial neurological improvement, as opposed to the success of hyperbaric oxygen therapy in the 2 patients in whom the delay was minimal. We conclude that as soon as the proposed open heart operation has been completed and there is an indication that air embolism has occurred, the patient should be treated with hyperbaric oxygen as quickly as possible, even before neurologic manifestations of cerebral ischemia appear.

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Arterial air embolism during cardiopulmonary bypass can result in serious brain damage or fatality. The incidence of this complication is estimated to be 0.1% [1, 2]. Currently more than 3,500 operations with cardiopulmonary bypass are performed annually in Israel, so that 3 patients with air embolism may be expected. Despite improvements in equipment and technology, these accidents still occur, demanding quick and accurate diagnosis and efficient therapeutic measures.

The immediate management includes Trendelenburg position, hypothermia, steroids, anticoagulants, oxygen, and volume expansion [3, 4]. Retrograde perfusion has been suggested as a means of decreasing the amount of air obstructing the cerebral circulation [5, 6], yet only 47% of the gas was recovered by this modality in an animal model [4]. The definite and specific treatment of this complication is hyperbaric oxygen (HBO). It has been concluded that every heart surgeon should ascertain the nearest hyperbaric facility and should adopt a contingency plan to be followed in the event of air embolism [7, 8].

We report 6 patients referred to our institute since January 1985 because of arterial air embolism during cardiopulmonary bypass. In each case diagnosis was made by the referring surgeon, based on his suspicion that a considerable amount of air had entered the systemic circulation during the operation. The patients were treated in a large multiplace hyperbaric chamber. The chamber is the only hyperbaric referral facility in Israel, and so these 6 patients reflect the total national clinical experience.

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Case Summaries

Six patients were referred with a diagnosis of air embolism. Of the 4 patients who had undergone coronary artery bypass grafting, 2 had undergone additional surgical procedures. The fifth patient had undergone closure of an atrial septal defect, whereas aortic valve replacement had been carried out in the sixth.

Two of the patients were referred immediately on completion of the operation, which made it possible to commence HBO therapy with a delay of only 2 to 3 hours. These 2 patients made a complete recovery. In the remaining 4 cases, the surgeons preferred to wait and observe the impact of the embolism on the recovery process. Only when normal recovery was not observed the following day were these patients referred for HBO. This resulted in a considerable delay in the commencement of therapy. Two of the 4 patients died, 1 was left with severe neurological impairment, and 1 was left with a mild motor deficit in one leg. In 5 cases, a modified version of the US Navy treatment table 6A was used (Fig 1). One patient was given 90 minutes of oxygen at 2.8 atmospheres absolute. The main clinical data are summarized in Table 1.

Comment

Arterial air embolism accompanying cardiopulmonary bypass may result in disability or even death. In a survey of 349 cardiac surgeons [1], the incidence of arterial air embolism was estimated to be 0.1%, but its true incidence seems to be higher [7]. The vast majority of instances are unreported and quietly ignored, mainly due to a fear of litigation.

According to the statistics of Stoney and associates [1], there were 429 instances of arterial air embolism in 374,819 cardiopulmonary bypass operations (0.11%). Of these, 61 resulted in disability (14.2%), and 92 patients

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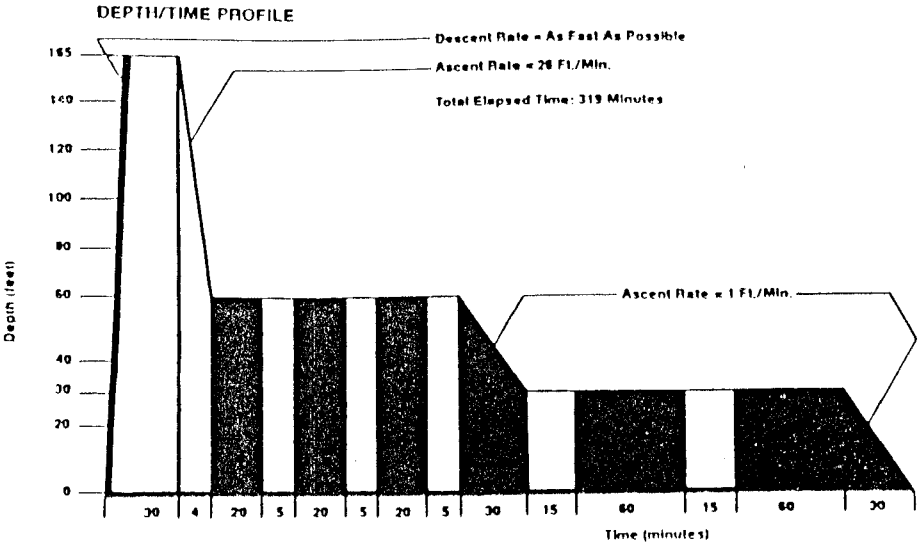

Fig 1. US Navy Treatment Table 6A. This graph indicates the pressure (depth in feet) inside the hyperbaric chamber at any given time, the rate at which pressure is increased or reduced, and the gas breathed during the various stages of the treatment. Solid areas denote intervals of exposure to pure oxygen, and open areas intervals of exposure to air. The table is used by us in a modified form. To increase the oxygen partial pressure during the first 30 minutes of therapy, a 50%/50% nitrogen/oxygen mixture is introduced instead of air at this critical stage.

(21.4%) died of the complication, which can occur at the onset of perfusion, during perfusion, or on resumption of cardiac function [7].

A number of causes of air entry into the bypass circuit have been documented, including rupture of arterial lines and connectors, oxygenator defects, arterial reservoir run dry due to insufficient venous return, and sudden acceleration of a roller pump [9, 10].

The control of air embolism lies in prevention. It is incumbent upon each open heart team to be prepared to deal with the situation should it arise. Guidelines for this and other emergency procedures should be established and practiced by the perfusionist team. The management of gross air embolism should include the following [11]:

1. The arterial pump is immediately stopped and the venous line clamped.
2. The patient is placed in a steep head-down position.
3. The aortic cannula is removed and the arterial circuit purged of air.
4. The arterial cannula is connected to the venous line to facilitate retrograde perfusion. Alternatively, the atrium can be cannulated separately.
5. Retrograde perfusion at a flow of 1 to 2 L/min and at a temperature of 20°C is carried out with the flow directed up the superior vena cava. This is continued for a few minutes with the suction system being used to retrieve blood exiting from the aortotomy.
6. The anesthetist exerts pressure on the carotid arteries, and retrograde perfusion is continued until the arterial system is cleared of air. In extreme cases, retrograde perfusion through the inferior vena cava may also be required.
7. Standard bypass is resumed with hypothermia, and attention is turned toward removing any air that may have entered the coronary system, by pharmacological

Table 1. Main Clinical Data

Patient No.	Age (y)	Sex	Operation	HBO Therapy	Time to HBO (h)	Main Symptoms	Outcome
1	53	M	CABG	6A[a]	17	Coma; mechanically ventilated; no response to pain; hypotonia	No response; severe impairment
2	62	F	CABG + carotid endarterectomy	2.8 ATA, 90 min	18	Coma; mechanically ventilated; hypotonia	No response; died
3	57	M	CABG + mitral valve replacement	6A[a]	20	Coma; mechanically ventilated; responsive to pain	Improved
4	4	F	Closure of ASD	6A[a]	2	Coma; mechanically ventilated	Complete recovery
5	55	M	CABG	6A[a]	20	Coma: mechanically ventilated; no response to pain; pupils dilated	No response; died
6	70	F	Aortic valve replacement	6A[a]	3	Coma; mechanically ventilated; pupils reactive to light	Complete recovery

[a] Modified

ASD = atrial septal defect; ATA = atmospheres absolute; CABG = coronary artery bypass grafting; F = female; HBO = hyperbaric oxygen; M = male.

elevation of the perfusion pressure and small-needle puncture of the distal coronary system.
8. The operative cardiac procedure is completed as quickly as possible.

Rewarming is carried out slowly to a core temperature of 35°C, and bypass is completed in the usual fashion. The patient is ventilated on 100% oxygen. The value of steroid therapy is unproven, but it may be beneficial. Hyperosmolar and diuretic therapy is mandatory and ideally should be monitored with intracranial pressure.

Hyperbaric oxygen is considered a specific therapy for air embolism because it is based on the mechanical compression of air bubbles according to Boyle's law: the volume of a gas is inversely proportional to the pressure to which it is exposed [8, 12]. Hence compression to 6 atmospheres absolute will reduce the size of an intravascular bubble to one sixth of its original volume, which is 55% of its original diameter. This reduction helps relieve the vascular obstruction and restores perfusion. Concomitantly, HBO reduces the blood nitrogen partial pressure, increases the gradient between the bubbles and the blood, and accelerates bubble resorption. In addition, HBO both increases ischemic brain tissue oxygenation and reduces brain edema by vasoconstriction [7].

Winter and colleagues [13] and Tomatis and co-workers [8] indicated that HBO therapy may be lifesaving even after a delay as great as 11 hours, before irreversible brain damage. Mader and Hulet [14] predicted that HBO therapy may be useful in the treatment of cerebral air embolism even if this therapy is delayed more than 24 hours after embolization. This is in accord with our experience [15].

Because minor air embolism is so common and the immediate clinical impact seems, erroneously [16], to be negligible, the cardiac surgeon is reluctant to refer the patient for HBO therapy, even when a major air embolus is seen entering the arterial circulation. Only when the patient remains comatose some hours later is HBO therapy considered.

Because our chamber is the only hyperbaric referral facility in Israel, our experience represents the total number of patients referred for HBO therapy since 1985. According to statistics from the Israel Ministry of Health, in the course of these 6 years 16,332 operations with cardiopulmonary bypass were performed. According to Stoney and associates [1] the estimated number of patients with air embolism during this time would be 16. We believe, then, that the 6 patients described reflect only a fraction of the patients who should have been referred for HBO. It might be mentioned that ground transportation using a mobile intensive care unit proved safe in all 6 cases, and caused no further morbidity. The multiplace chambers in most hyperbaric centers can offer continuous intensive care during HBO therapy, as mechanical ventilators and monitoring equipment are available.

Definite conclusions cannot be drawn from the 6 patients described, but of the 4 patients in whom HBO therapy was delayed for 17 to 20 hours, only 1 showed significant neurologic improvement (patient 3), as opposed to the success of HBO therapy in patients 4 and 6, in whom the delay was minimal.

We conclude that massive arterial air embolism should be treated with HBO as quickly as possible, even before neurologic manifestations of cerebral ischemia appear.

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