11 Cerebral Air Embolism

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HBO is the most effective treatment of air embolism; it reduces the size of air bubbles and counteracts the secondary effects. This chapter on cerebral air embolism examines:

Causes

The introduction of air into the venous or the arterial system can cause cerebral air embolism leading to severe neurological deficits. The first known recognition of arterial air embolism was reported by Morgagni in 1769, and later, in 1821, Magendie described the consequences of pulmonary overinflation leading to arterial gas embolism. The most common causes reported in the literature are iatrogenic, the embolism occurring as a result of invasive medical procedures or surgery. Less commonly, air embolism occurs in divers undergoing rapid decompression and in submarine escape. Causes of air embolism are shown in Table 11.1.

Table 11.1
Causes of Air Embolism

A. Sudden decompression or ascent in diving and submarine escape
- Pulmonary barotrauma – "burst lung" in divers
- Rapid decompression in an altitude chamber for flight training

B. Trauma
- Cardiopulmonary resuscitation in patients with undetected lung injury
- Head and neck injuries
- High-altitude accidents

C. Iatrogenic
1. Diagnostic and minor procedures
- Intravenous fluids and central venous pressure (CVP) lines
- Arterial lines for blood and medication infusion
- Angiography: diagnostic and therapeutic catheterization of blood vessels
- Mechanical positive pressure ventilation
- Air contrast salpingogram
- Air insufflation with pneumatic otoscope
- Needle biopsy of the lung
- Hemodialysis
- Gastrointestinal endoscopy

2. Intraoperative complications
- Neurosurgical operations in the sitting position: tear into veins in the posterior fossa or the cervical spinal canal
- Cardiac surgery: open heart surgery with extracorporeal circulation
- Vascular surgery: carotid endarterectomy with shunt
- Thoracic surgery: opening of pulmonary veins at subatmospheric pressures
- Endobronchial resection of lung tumor using Neodymium-YAG laser
- Pelvic surgery in Trendelenburg position. Operative hysteroscopy with laser
- Cesarian section

D. Miscellaneous and rare causes
- Faulty abortion
- Orogenital sex during pregnancy
- Inhalation of helium directly from a pressurized helium tank.
- Ingestion of hydrogen peroxide solution

There are approximately 20,000 cases of air embolism per year in the USA. The exact incidence of various causes is difficult to determine, as not all cases are reported in the literature. An excellent review of the topic is presented elsewhere (Hodics & Linfante 2003). Some victims recover spontaneously. This review concerns those cases where hyperbaric treatment was used.

The incidence of air embolism during cardiopulmonary bypass operations is 0.1%. The actual prevalence may be higher because several such complications are not recognized and reported. Air enters the venous system in 30%–40% of the patients undergoing neurosurgical operations in the sitting position. Air embolism can occur during neuro-angiographic interventional procedures such as aneurysm coiling embolization and carotid stent placement but overall incidence during diagnostic neuroangiographic procedure is very low in the order of 0.08% (Gupta *et al* 2007).

Mechanisms

In iatrogenic cases the air is either sucked into the veins with negative pressure or introduced into the veins or arteries under pressure. The lung is usually an effective filter for air bubbles greater than 22 μm in diameter when air is injected slowly. A bolus injection of air more than 1.5–3 ml/kg exceeds the filter capacity of the lungs and produces embolization through the left heart into the arterial circulation until it blocks arterioles 30–60 μm in diameter. Air has a large surface tension at the blood-air interface and the globules of air cannot be deformed enough to navigate the capillaries.

A patent foramen ovale occurs in 20% to 35% of the normal adult population and in one out of ten of these is at risk of having arterial air embolism when air enters the venous system inadvertently. The exact prevalence rate of functional right to left atrial shunt in the healthy adult population, however, is unknown. In the absence of such a shunt, venous air must first traverse the pulmonary vasculature in order to enter the cerebral circulation.

In pulmonary barotrauma, lung volumes expand during rapid ascent. When alveolar pressure exceeds 80–100 mmHg, air can be forced into pulmonary capillaries. Alveoli may rupture into the pleural space, causing pneumothorax, or into the pulmonary veins, where the embolus may traverse the left side of the heart to enter the aorta and may ascend the carotid arteries to the cerebral circulation, as the diver is usually upright during ascent.

Pathophysiology

Air emboli lodge distally in the smaller arteries and arterioles of the brain and obstruct the flow of blood. The

result is ischemia, hypoxia, and cerebral edema. Even when the bubble is dissolved, a "no reflow" phenomenon may occur in the damaged tissues. The bubble acts as a foreign body and starts a number of biochemical reactions in the blood. Platelets are activated and release vasoactive substances including prostaglandins. The bubble may damage the endothelial cells of the vessel wall by direct contact. Margination and activation of leukocytes follows, and may cause a secondary ischemia. If the bubble persists, it is surrounded by platelets and fibrin deposit, which may prevent dislodging of the bubble. Although the potential of large bubbles to cause cerebral injury is not disputed, there is controversy over the significance of exposure to small bubbles in cardiac surgery. It is known that postsurgical neuropsychological deficits do correlate positively with the number of emboli to which patients are exposed; to date, however, the technology for distinguishing between gaseous and particulate emboli or for sizing emboli accurately is not readily available (Mitchell & Gorman 2002).

Air bubbles injected directly into the cerebral circulation of experimental animals can open the blood-brain barrier. The barrier, however, repairs itself within a few hours. Ischemic hypoxia produced by air embolism is not severe enough to produce gross cerebral infarction, but produces necrosis of the deep cortical layers at the gray-white matter junction.

There may be segmental arterial vasospasm followed by dilatation, and some of the air may escape from the arteries into the veins via capillaries. As a result of arterial obstruction regional cerebral blood flow (rCBF) declines and EEG activity may cease in the affected region. The changes are typical of cerebral ischemia, but the blood-brain barrier permeability increases immediately after air embolism, in contrast to vascular occlusion from other causes, where the onset is delayed. Focal ischemia of short duration does not lead to cell loss, and the processes causing deterioration are potentially reversible. The other potentially reversible process that occurs in the tissues is cerebral edema. Although the brain is the major concern in arterial embolism, the coronary arteries may occasionally be involved.

Animal brain models of cerebral arterial gas embolism may be useful in comparing the effectiveness of various recompression schedules. Murrison (1993) has reviewed various animal models for neurophysiological investigations of pathophysiology of central nervous system (CNS) damage in arterial gas embolism. Most of these studies involve injection of air into cerebral vasculature. Secondary CNS deterioration may be may be due to endothelial damage or change in blood constituents rather than mechanical bubble action and may explain the failure of recompression therapy in such cases. The results of these animal studies cannot be extrapolated to humans.

Clinical Features

Clinical manifestations, essentially neurological or cardiovascular disorders vary greatly. The clinical features of air embolism depend on the patient's posture, the route of entry of air, the volume of air, the size of the bubbles, and the rate of entry of air. If the patient is reclining, air is more likely to enter the coronary arteries, whereas it is more likely to enter the cerebral arteries if the patient is upright. Neurological sequelae have been estimated to occur in 19 to 50% of the patients with cerebral air embolism.

Signs and symptoms of air embolism in divers may not be clear-cut. In 50% of such cases, dysbaric air embolism was found to be part of dysbarism syndrome including decompression sickness. A sudden change in sensorium is the most common symptom and ranges from disorientation to coma. Focal neurological deficits such as hemiplegia or monoplegia may occur, according to the location of the lesions. Respiratory arrest and seizures are less common. A shock-like state may occur with massive embolism. Associated symptoms may be those of pneumothorax (in pulmonary barotrauma) or myocardial ischemia. Liebermeister's sign, i.e., the presence of areas of pallor on the tongue after air embolism, may be found.

Diagnosis

The diagnosis of air embolism is based on a careful consideration of the patient's history and neurological findings. In cases of sudden decompression with neurological deficit, the diagnosis is easier. During surgical procedures monitored by doppler ultrasound, air embolization is detected at an early stage and appropriate measures can be taken to stop further air entering the blood vessels. Transcranial doppler studies show that microscopic cerebral artery air emboli are present in virtually all patients undergoing cardiac surgery. Microbubbles can be detected with two-dimensional echocardiography, which is often used for this purpose during open heart and bypass surgeries (Kearney *et al* 1997). EEG monitoring is also useful for early detection of acute cerebral dysfunction.

Subtle changes in mental function may be a major manifestation even in the absence of other objective neurological signs. CT scan offers a possibility of diagnosis of subclinical lesions of the brain. Air bubbles may also be seen on fundoscopic examination. Air may be seen in the cerebral arteries during a neurosurgical operation, or air can be demonstrated in a specimen of arterial blood. A high index of suspicion is very important in diagnosis. Under suspicious circumstances air embolism should be assumed present unless otherwise proven. In some cases the diagno-

sis is proven only after successful response to hyperbaric therapy.

In air embolism associated with diving, there is muscle injury and elevated serum creatine kinase which a marker of the severity of this complication (Smith & Neuman 1994).

Treatment

Emergency measures include administration of 100% oxygen, using a closely fitting mask, and transport of the patient to a hyperbaric facility. If transport by air is unavoidable, the patient should travel in a pressurized cabin, and the aircraft should stay at a low altitude. A bolus dose (10 mg) of dexamethasone may be given to prevent cerebral edema. Oxygen serves to reduce the size of the air bubble by depletion of nitrogen and also counteracts the hypoxia and ischemia of the surrounding brain tissue.

The important consideration in treatment of cerebral air embolism is preparedness and anticipation. Procedures with a known risk of air embolism should not be performed far away from a hyperbaric facility, and a hyperbaric chamber should be available in institutions that conduct open heart surgery. Time is the more important element in management – the shorter the delay, the better the outcome. Emboli large enough to produce symptoms require immediate treatment because of the risk of "gas lock" in the right side of the heart and subsequent circulatory failure (Jørgensen *et al* 2008).

The generally accepted treatment of air embolism is immediate compression to 6 ATA air for a period of not more than 30 min followed by ascent to 2.8 ATA with oxygen. The rationale of this approach is as follows:

1. Compression of the bubbles reduces their size. According to Boyle's law, the volume of a gas is inversely proportional to the pressure exerted on the gas. Compression to 6 ATA would reduce the size of a bubble to one-sixth, or approximately 17%, of its original size (Table 11.2). The reduction of the surface area of the bubble to 30% reduces the inflammatory effect of the bubble-blood interface.

Table 11.2
Relative Volume and Surface Area of a Bubble with Compression

Pressure (ATA)	Relative volume (%)	Relative surface area (%)
1	100	100
2.8	35	50
6	17	30

2. Delivery of high levels of oxygen is important to counteract the ischemic and hypoxic effects of vascular obstruction. Breathing oxygen (100%) at 2.8 ATA leads to an arterial pO_2 level of 1800 mmHg. At this pressure 6 ml oxygen is dissolved in 100 ml plasma.
3. Fick's law can be applied to relate the rate of nitrogen diffusion to the concentration gradient between the bubble and the surrounding tissue. Oxygen at 100% concentration improves the diffusion of nitrogen from the bubble.
4. Cerebral edema associated with cerebral air embolism is decreased by HBO.
5. Vasoconstriction induced by HBO inhibits air embolus redistribution. This is possible because the reactivity of the cerebral arteries is not impaired in cerebral air embolism (Gordman & Browning 1986).

The first experimental study employing hyperbaric therapy was conducted by Meijne *et al* (1963). They injected air into the carotid arteries of rabbits and showed a remarkable improvement in the survival rate of the animals treated with HBO. Leitch *et al* (1984a–d) carried out a series of experimental studies to reassess the hyperbaric treatment of air embolism. They tested the question, "Is there a benefit in beginning treatment at 6 ATA?" in dog models of air embolism treated at various pressures. The effectiveness of the treatment was assessed by sensory evoked potentials (SEP) and CBF. It was concluded that there was no advantage in using air at 6 ATA prior to treatment with oxygen at 2.8 ATA. The data showed that clearance of air is probably independent of pressure past the threshold of 2 ATA and is certainly hastened by oxygen. Approximately 8 min were required to clear the embolism. A number of air-treated dogs had redistribution of air embolism after initial decompression and concomitant reduction of CBF. More recently, McDermott *et al* (1992) carried out a study of various pressure schedules in experimental feline arterial air embolism with assessment of severity by cortical SEP. They found no additional benefits of initial treatment at 6 ATA as compared to 2.8 ATA.

Transcranial doppler studies show that microscopic cerebral artery air emboli (CAAE) are present in virtually all patients undergoing cardiac surgery. Massive cerebral arterial air embolism is rare. If it occurs, HBO is recommended as soon as surgery is completed. Dexter and Hindman (1997) have used a mathematical model to predict the absorption time of air embolus, assuming that the volumes of clinically relevant air emboli vary from 10^{-7} to at least 10^{-1} ml. Absorption times are predicted to be at least 40 h during oxygenation using breathing gas mixtures of fraction of inspired oxygen approximately equal to 40%. When air emboli are large enough to be detected by CT, absorption times are calculated to be at least 15 h. Decreases in cerebral blood flow caused by the air emboli would make the absorption even slower. Analysis of these authors suggests that if the diagnosis of massive

Table 11.3
Examples of Applications of HBO for Cerebral Air Embolism

Authors and year	No. of cases	Cause	Pressure used	Results and comments
Davis *et al* (1990)	1	Cesarian section	Table 6	HBO treatment started 8 hours after onset with impairment of consciousness and left hemiplegia. Recovered with minimal neurological deficit.
Arnon *et al* (1991)	1	Open heart surgery	Table 6A	HBO treatment was started 30 hours after the incident with coma, decerebrate rigidity and seizures. Recovered with minimal residual neurological deficits at 14 months follow-up.
Kol *et al* (1993)	6	Cardiopulmonary by-pass	Table 6A	2 died 2 partial recovery 2 full recovery
Rios-Tejada *et al* (1997)	1	Decompression at flight level 280 (28,000 ft) in an altitude chamber.	Table 6A (extended) +3 HBO sessions at 2 ATA/90 min	Complete recovery from left hemiplegia.
Droghetti *et al* (2002)	1	Paradoxical air embolism during percutaneous nephrolithotripsy in the prone position.	Table 6	Patient presented neurological deficits 8 hours later, when HBO treatment was started. Full recovery.
Wherrett *et al* (2002)	1	Diagnostic bronchoscopy in a patient with previous lobectomy for bronchogenic carcinoma.	Modified Table 6	Treated 52 h after the event. Discharged after fully recovery 1 week later.

CAAE is suspected, CT should be performed, and consideration should be given to HBO therapy if the emboli are large enough to be visualized, even if patient transfer to a HBO facility will require several hours.

Some authors recommend supportive care as the primary therapy for venous gas embolism, while HBO therapy in addition to supportive care is the first line of treatment for arterial gas embolism (Fukaya & Hopf 2007). The criterion for use of HBO is clinical manifestation, particularly neurological and not the source of air embolism.

Clinical Applications of HBO

Clinical applications of HBO for air embolism during the past decade are shown in Table 11.3. If we consider the overall mortality of air embolism without hyperbaric treatment as 30%, these results represent a remarkable improvement. A controlled prospective study has shown that mortality can be reduced to 14% if hyperbaric oxygen therapy is given within 12 hours of the accident (Bacha *et al* 1996). Treatment appears to be ineffective after irreversible damage has already been done.

HBO has been used successfully in cases of air embolism as a complication of open heart surgery, endoscopy (Raju *et al* 1998) and transthoracic percutaneous thin-needle biopsy (Regge *et al* 1997). The usual schedule of hyperbaric treatment is US Air Force Modification of US Navy Table 6 (Figure 10.5; Chapter 10). The initial approach is to compress the patient to 6 ATA. After 30 min, decompression is carried out to 2.8 ATA.

Ancillary Treatments

The following treatments have been used in addition to hyperbaric therapy.

Antiplatelet Drugs. These have been used to counteract the platelet aggregation associated with air embolism. The use of heparin as anticoagulant is considered risky due to the danger of hemorrhage in infarcted areas. Patients who are already on heparin have a better prognosis after air embolism than those who are not anticoagulated. This is particularly noted during cardiopulmonary bypass for cardiac surgery. Oral aspirin is safer but takes about 30 min to act after ingestion.

Steroids. These have been used to prevent cerebral edema. Delayed cerebral edema can occur after initially good results from recompression following air embolism. Steroids should be administered cautiously during HBO as they may accelerate the development of oxygen toxicity.

Hemodilution. Hematocrit has been shown to have a relation to the infarct size in vascular occlusion, and many cases of air embolism display hemoconcentration. Hemodilution, e.g., by dextran-40, is indicated. Lowering of hematocrit also causes a reduction in oxygen-carrying capacity, but this is more than adequately compensated by HBO.

Control of Seizures. An anticonvulsant medication may be required for control of seizures. Prophylactic use of lidocaine not only controls seizures but also reduces infarct size and prevents cardiac arrhythmias associated with air embolism.

Measures to Improve Cerebral Metabolism. Loss of blood supply causes immediate reduction of neuronal pools and increased production of lactate. The total energy available is reduced. Increased blood glucose levels are associated with increased lactate production by the ischemic brain and increase in infarction. The control of blood glucose, therefore, is important after air embolism and routine use of intravenous dextrose should be avoided. There is evidence that HBO serves to normalize the cerebral metabolism (see Chapter 17) and also lowers blood glucose.

Hyperbaric Treatment in Special Situations

Cerebral Edema

The following example is given to illustrate the special use of HBO in cerebral edema. Thiede and Manley (1976) reported a patient with air embolism who responded to initial compression to 6 ATA but deteriorated into coma and decerebrate posturing during decompression at 1.9 ATA. There was increased intracranial pressure, indicating cerebral edema. The patient was given repeated HBO treatments twice daily at 2.8 ATA (100% oxygen) and recovered completely. HBO in this case was doubly indicated – for air embolism as well as for cerebral edema.

Cardiovascular Surgery

Calverley *et al* (1971) reported air embolism during cardiac catheterization in a 4-month-old infant with ventricular septal defect. A quantity of 10 ml air was inadvertently injected into the right ventricle. Anesthesia was terminated and air compression was done to 6 ATA 35 min after the episode. Decompression was started after a further 15 min and completed in 5 h. No oxygen was given. The infant made a good recovery and the planned cardiac surgery was carried out. Calverley *et al* made an important observation about nitrous oxide anesthesia: if air embolism occurs during this type of anesthesia, nitrous oxide diffuses rapidly into enclosed pockets of gas, causing an increase in pressure (or an increase in volume if the surrounding tissues permit). The authors recommended that nitrous oxide anesthesia should be discontinued if air embolism occurs.

HBO has been used to treat patients with extensive neurological deficits from air emboli during open heart surgery. Treatment is usually not started until after completion of surgery, but is still effective. Some complicated operations in cardiac surgery and neurosurgery cannot be aborted because of air embolism. In such cases, compression treatment can be started after completion of the operation.

Huber *et al* (2000) reported a case of a 5-year-old girl who suffered a massive arterial air embolism during surgical closure of an atrial septal defect. They successfully treated a proven arterial air embolism with intraoperative (retrograde cerebral perfusion) combined with postoperative procedures (deep barbiturate anesthesia and HBO). At discharge the girl had fully recovered from the initial neurologic defects.

Hypothermia has been used in cardiac surgery for cerebral protection. Patients who suffer massive air embolism during cardiopulmonary bypass can be treated by using a combination of hypothermia and HBO with good results. According to Charles' law, the volume of a gas varies according to temperature. Theoretically hypothermia is expected to decrease the size of the gas bubbles and should be beneficial in air embolism. Animal experimental studies are required to determine if HBO and hypothermia complement one another in air embolism.

Neurosurgery

Air embolism following posterior fossa surgery in the sitting position can be promptly treated by recompression according to the standard schedule. The patients usually recover without neurological deficits.

Pulmonary Barotrauma

Leitch and Green (1987) reviewed treatment of 89 cases of air embolism due to pulmonary barotrauma in divers. There was a 65% success rate with hyperbaric treatments, and one of the victims had a relapse. The authors concluded that although most cases would recover with oxygen at 2.8 ATA, there was no reason to alter the established technique of initial compression with air to 6 ATA prior to HBO at 2.8 ATA. Air embolism associated with pulmonary barotrauma during rapid decompression in an altitude chamber has been managed by the use of treatment table 6A (Rudge 1992).

Pulmonary barotrauma with air embolism has been reported as a complication of HBO therapy for a non-healing ulcer of the foot (Wolf *et al* 1990). Pneumothorax has been reported as a complication of recompression therapy for cerebral arterial gas embolism associated with diving (Broome & Smith 1992).

In an unusual presentation of cerebral air embolism, a patient became unresponsive and developed subcutaneous emphysema during the direct insufflation of oxygen into the right middle lobe bronchus (Wherrett *et al* 2002). An endotracheal tube and bilateral chest tubes were immediately placed with resultant improvement in the oxygen saturation. However, the patient remained unresponsive with

extensor and flexor responses to pain. Later, there was seizure activity requiring anticonvulsant therapy. CT scans of the head and cerebral spinal fluid examination were negative, though the electroencephalogram was abnormal. A CT of chest showed evidence of barotrauma. 52 hours after the event, a presumed diagnosis of cerebral air embolism was made, and the patient was treated with HBO using a modified US Navy Table 6. 12 hours later he had regained consciousness and was extubated. He underwent two more HBO and was then discharged from hospital 1 week after the event, fully recovered. Although HBO was started after significant delay, the patient made a good recovery.

Air Embolism During Invasive Medical Procedures

Catron *et al* (1991) used HBO to treat two patients with cerebral air embolism resulting from invasive medical procedures. Both patients recovered without any evidence of damage on clinical examination and MRI.

Cerebral Air Embolism During Obstetrical Procedures

Two cases of cerebral air embolism occurring during cesarian section were treated successfully with the use of HBO (Sadan *et al* 1991; Davis *et al* 1990). Air embolism manifested by cortical blindness was reported in a patient following induced abortion by means of intra-amniotic hypertonic saline instillation and the patient made a complete recovery after treatment with HBO (Weissman *et al* 1989). Venous air embolism is likely during cesarian section as air enters uterine sinuses, particularly if the placenta separates before delivery as in the case of placenta previa (Davis *et al* 1990).

Cerebral Air Embolism Due to Orogenital Sex During Pregnancy

Twelve cases of this type have been reported in the literature; only one of them survived. Two cases have been treated successfully using HBO (Bray *et al* 1983; Bernhardt *et al* 1988).

Cerebral Embolism Due to Hydrogen Peroxide Poisoning

Hydrogen peroxide can produce acute gas embolism. There is a case report of an adult who suffered an apparent stroke shortly after an accidental ingestion of concentrated hydrogen peroxide (Mullins & Belltran 1998). Complete neurologic recovery occurred quickly following HBO treatment. In another case report, a patient developed cerebral air embolism a short time after ingestion of a small amount of hydrogen peroxide manifested by hematemesis, left sided hemiplegia, confusion, and left homonymous hemianopsia. Initial laboratory studies, chest x-ray, and brain CT were normal. MRI demonstrated areas of ischemia and 18 h hours after arrival, the patient underwent HBO treatment with complete resolution of symptoms (Rider *et al* 2008). Of the seven reported cases of air embolism from hydrogen peroxide that did not undergo HBO, only in one patient was there a report of symptom resolution. HBO can be considered as the definitive treatment for gas embolism from hydrogen peroxide ingestion as with all other causes of acute gas embolism.

Pediatric Air Embolism

Van Rynen *et al* (1987) reported successful treatment of cerebral air embolism in a 3-month-old infant who had undergone a palliative closed heart operation. The treatment was conducted in a Reneau monoplace chamber using initial recompression to 6 ATA. The infant made a good recovery.

Relapse Following Spontaneous Recovery

In cases of spontaneous redistribution of air bubbles, a period of apparent recovery is frequently followed by relapse. The etiology of relapse appears to be multifactorial and is chiefly the consequence of a failure of reperfusion. Prediction of who will relapse is not possible, and any such relapse carries poor prognosis. It is advisable, therefore, that air embolism patients who undergo spontaneous recovery be promptly recompressed while breathing oxygen (Clark *et al* 2002). Therapeutic compression serves to antagonize leukocyte-mediated ischemia-reperfusion injury; to limit potential re-embolization of brain blood flow, secondary to further leakage from the original pulmonary lesion or recirculation of gas from the initial occlusive event; to protect against embolic injury to other organs; to aid in the resolution of component cerebral edema; to reduce the likelihood of late brain infarction reported in patients who have undergone spontaneous clinical recovery; and to prevent decompression sickness in high gas loading dives that precede accelerated ascents and omitted stage decompression.

Delayed Treatment

Air embolism should be treated as quickly as possible after it is detected. This is not always possible and several cases receive delayed treatment. HBO treatments have led to re-

covery in cases of air embolism with severe neurological deficit where treatment was delayed for 24 h. In a subgroup of 5 patients with air embolism secondary to cardiopulmonary bypass accidents, pulmonary barotrauma induced by mechanical ventilation and central vein catheterization, significant recovery was noted even when treatment was started 15 to 60 h after the event (Bitterman & Melamud 1993). Full recovery was reported in a case of hemodialysis associated venous air embolism, where HBO treatment was commenced 21 h after the event when the patient already appeared to be decerebrate (Dunbar *et al* 1990).

Conclusions

Hyperbaric treatment has been proven to be unquestionably indicated for the treatment of air embolism with neurological deficits. The conventional methods of treatment, such as posturing the patient in certain ways, aspirating the air, providing normobaric oxygen, closed chest massage, and steroids, have not been adequate to manage this problem. The consensus concerning the pressure favors the retention of 6 ATA initial compression with air. If the patient's condition does not permit exposure to this high pressure or the chamber immediately available cannot provide this pressure, 100% oxygen at 2.8 ATA would be acceptable as an alternative, particularly when only a monoplace chamber is available. The diagnosis of air embolism cannot always be made with certainty. There is need to improve technologies for early detection of air bubbles. It is acceptable to treat the patient with compression if air embolism is suspected, and the response to compression may be diagnostic in such cases. Early treatment provides better results than late treatment but HBO treatment should be considered at any stage the patient presents.

Considering that air embolism is a complication of medical and surgical procedures, it stands to reason that hyperbaric chambers should be available at clinics that perform such procedures. Open heart surgery, certainly, should not be done in a hospital that does not have a hyperbaric chamber.